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OLIVER WENDELL HOLMES AND PUERPERAL FEVER*

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BOSTON

ON THE evening of February 13, one hundred years ago, the speaker before the Boston Society for Medical Improvement had reached the conclusion of his essay "The Contagiousness of Puerperal Fever." He was a small, frail man just about to enter middle age — for he was thirty-four years old — and his name was Oliver Wendell Holmes. His face had lost much of its customary expression of humorous benignity, his sensitive mouth was firm, and the usual twinkle was gone from his eyes; for never had he been so much in earnest. He said:

> There is no tone deep enough for regret, and no voice loud enough for warning. The woman about to become a mother, or with a newborn infant upon her bosom, should be the object of trembling care and sympathy wherever she bears her tender burden, or stretches her aching limbs. The very outcast of the streets has pity upon her sister in degradation when the seal of promised maternity is impressed upon her. The remorseless vengeance of the law, brought down upon its victim by machinery as sure as destiny, is arrested in its fall at a word which reveals a transient claim for mercy. The solemn prayer of the liturgy singles out her sorrows from the multiplied trials of life, to plead for her in the hour of peril. God forbid that any member of the profession to which she trusts her life, double precious at that eventful period, should hazard it negligently, unadvisably, or selfishly!

You need no aid from me to place America's most charming writer in the world of letters, but perhaps you will permit me to recall his position in the Boston medicine of his day and to remind you of his contribution to the arrest of the fearsome pestilence that had needlessly destroyed the lives of so many women in childbed. And perhaps, also, you will allow me to express, however imperfectly, the peculiar affection that his memory inspires in all Boston doctors, and in every student in Harvard Medical School. As professor of anatomy, he evoked a legend that is as fresh today as if he had just bade his final class farewell, and, clutching silently its gift — a silver loving cup — had descended for the last time those thirty-two well-worn steps that he had climbed for five and thirty years to instruct, amuse, and delight two generations of his pupils.

When Holmes read his paper on puerperal fever his medical contemporaries knew him as a witty dinner companion, the author of a volume of facile and melodious verse, a well-trained and intelligent physician, and a none-too-successful practitioner. On graduation from Harvard College, after a brief and boring experience with the law, he entered Harvard Medical School, or the Massachusetts Medical College, as it was then called. In 1833 he left medical school temporarily and went to Europe where he spent a little over two years at the University of Paris, then in a position of unchallenged pre-eminence. The great Louis, who originated the statistical study of disease, taught there in those days, as did such other famous men as Dupuytren, Lisfranc, and Velpeau. Of Baron Larrey, the chief surgeon of the Grande Armée, he[1] said, "To go around the Hôtel des Invalides with Larrey was to live over the campaigns of Napoleon, to look on the sun of Austerlitz, to hear the cannons of Marengo, to struggle with the icy waters of the Beresina, to shiver in the snows of the Russian retreat, and to gaze through the battle smoke upon the last charge of the red lancers on the redder field of Waterloo." There, also, he encountered Ricord, who first dis-

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tinguished between gonorrhea and syphilis and who had so little faith in morality that Holmes called him "the Voltaire of pelvic literature . . . who would have submitted Diana to treatment with his mineral specifics and would have ordered a course of blue pills for the Vestal Virgins."

Holmes returned to Boston in 1835, received his M.D. from Harvard in 1836, and immediately began practice. He won the Boylston Prize in 1836 and 1837, on the first occasion with an essay "The Indigenous Intermittent Fever of New England,"[3] — malaria — which is a medical classic. In 1838 he was appointed professor of anatomy at Dartmouth Medical School and held that chair for two years. With Jacob Bigelow he edited in 1839 the American edition of Marshall Hall's *Theory and Practice of Medicine*, and three years later he published his essay "Homeopathy and its Kindred Delusions,"[4] wherein he demolished by *reductio ad absurdum* the doctrines of infinitesimal dosage and *similia similibus curantur*. He was well established in Boston also as a teacher of medicine, for in 1838 with Jacob Bigelow, Edward Reynolds, and D. Humphreys Storer he founded the Tremont Street Medical School and taught auscultation and percussion, newly imported from Europe, and microscopic anatomy, another novelty in America. So successful was this school, which later gave summer courses to Harvard medical students, and so practical was its clinical teaching, that the university absorbed it in 1847. It is evident, therefore, that by 1843 Holmes not only had behind him an excellent professional education, but also had attained considerable importance as a medical writer and teacher.

When Holmes read his essay on puerperal fever, it is most unlikely that he had ever encountered in his own practice a case of that disease. Moreover, there was no large obstetric clinic in Boston where he could have studied it. At the Boston Lying-in Hospital, then housed in a small building on Washington Street, there were only about twenty-five women delivered each year — not enough material for even a small epidemic. Aside from the city almshouse, known euphemistically as the House of Industry, that was the only hospital where pregnant women were admitted. His interest in puerperal fever, therefore, must have been aroused in some other way. If we read, as Cutter did, the minutes of the Boston Society for Medical Improvement for the preceding fall, we find reports of two doctors and a medical student who had acquired infected hands at autopsies performed on two women dead of puerperal fever. The student and one of the doctors died; the other recovered after a long illness. At that time, also, there were small scattered epidemics in Boston and the neighboring towns, and a number of these cases had occurred successively in the practices of individual doctors. Such circumstances had led Dr. J. B. S. Jackson[4] to ask "the opinion of the Society as to the Contagion of Puerperal Fever, and the probability of Physicians communicating it from one patient to another." This suggestion, and these cases occurring in the practices of his colleagues, led Holmes, who had a broad knowledge of medical literature, to quote the authorities of the past. Charles White,[5] of Manchester, England, said he believed — like most of his predecessors and many of his contemporaries — that childbed fever resulted from retention of the lochia. In 1773, to encourage its escape, he invented "postural drainage," and he devised a jointed bed operated by a ratchet that was exactly like those we see today in every hospital ward; he also constructed a reclining chair to serve the same purpose when the patient got up. Moreover, White was the first to demand cleanliness in the lying-in chamber and to isolate infected patients from those who were well. In 1783, Pouteau,[6] Holmes noted, had detected the association of childbed fever with erysipelas, as had Robert Ceely[7] in 1831 and Thomas Nunnelley[8] in 1841. Alexander Gordon[9] had made in 1795 the first epidemiologic study of the disease, using as the basis an outbreak that had occurred in his city of Aberdeen; and he confessed to his great chagrin that in some cases he must have carried the contagion from one patient to another. Thomas Denman was well known to all who practiced midwifery. His tiny book of obstetrical aphorisms,[10] published at the turn of the nineteenth century, reposed in the tail-coat pocket of almost every British and American doctor, to be withdrawn and consulted secretly behind the woodshed when one was in a tight place. Denman[11] also said in 1801 that the disease might be carried from patient to patient, and he pointed out that foul bed linen and dressings might be the means by which this was effected. Finally, Holmes quoted Sir Thomas Watson,[12] his contemporary and the most distinguished English physician of his day, who only the year before had written: "Indeed, I believe that these cases of puerperal fever occurring in succession to the same practitioner are something more than ordinary contagion, operating through the medium of a tainted atmosphere. I believe them to be instances of direct inoculation."

Thus armed with the recent experiences of his colleagues and with the evidence drawn from his reading, Holmes announced his conviction that puerperal fever was carried from one patient to another, and that the person who conveyed the contagion was usually the physician or the nurse.

He evolved no theories about the nature of the contagion, nor did he speculate about the mode of its transmission. As he said later, "When facts are numerous, and unquestionable and unequivocal in their significance, theory must follow them as best it may, keeping time only with their step, and not go before them, marching to the sound of its own drum and trumpet." He drew up certain rules of guidance. These were: no doctor practicing midwifery must take an active part in an autopsy on a woman dying of puerperal fever; if, however, he should be present at such an autopsy, even though he did not touch the tissues, he must bathe thoroughly, change all his clothes, and allow twenty-four hours to pass before attending a woman in labor; he must adopt the same precautions after treating a case of erysipelas — he would be even wiser if he avoided the lying-in chamber as long as this contact existed; if a doctor had a case of puerperal fever, he should consider his next obstetric patient in danger of being infected by him; if two cases occurred in his practice, he must give up obstetrics for at least a month; and if he had three cases, he must consider them *prima-facie* evidence that he was responsible for the infection. Moreover, he warned, the doctor must inquire diligently concerning the nurses and attendants who take care of his patients; if they had been in contact with cases of childbed fever, they must not be employed. Finally he said, "A private pestilence in the practice of a physician is not a misfortune but a crime."

All of us who love Holmes could conceive no greater satisfaction than to hail him as the discoverer of the contagious nature of puerperal fever. But here, as so many times, cold facts and the printed word prevent, for he was not its discoverer, nor — as we have seen by the array of authorities he himself quoted to prove his point — did he ever claim to be. But his was the first voice raised in America to drive home this appalling truth; and never, in the whole history of medicine, did any advocate plead his case with greater eloquence or with more conviction.

Holmes's essay appeared in the April, 1843, issue of the *New England Quarterly Journal of Medicine and Surgery*, an obscure periodical that soon ceased publication. At the request of his friends, however, he secured reprints; and some of them eventually fell into the hands of Charles D. Meigs and Hugh L. Hodge, both of Philadelphia; had not this happened, the world might then have profited little by his essay, for it would have remained hidden in the files of a defunct medical journal. Thereupon both Meigs and Hodge expressed their unequivocal disapproval of Holmes, his essay, and the idea that doctors could carry infection from one patient to another. Hodge[13] was courteous and moderate in his statements, but Meigs[14] attacked the Bostonian in a most insulting fashion. "I prefer," said he sanctimoniously, "to attribute these cases to accident or Providence, of which I can form a conception, than to contagion, of which I cannot form any clear idea." From the eminence of his chair at the Jefferson Medical School he glared down at the little New England upstart, and he characterized the statements of Holmes and of those who held the same beliefs as the "jejune and fizenless dreamings of sophomore writers."

To defend his position, Holmes[15] republished his essay in 1855 under the title "Puerperal Fever as a Private Pestilence," and to it he added certain remarks. He was a kindly man and the soul of courtesy, but it is a pleasure to find that when he tried his hand at polite invective he was good at that, too. In noting that his opponents were professors in two of the country's leading medical schools, and that as such their opinions, although wrong, would have a profound effect on their students, he said that these young men "naturally have faith in their instructors, turning to them for truth and taking what they might choose to give them; babes in knowledge, not yet able to tell the breast from the bottle, pumping away for the milk of truth at all that offers, were it nothing better than a professor's shrivelled forefinger."

Charles Delucena Meigs was one of the most remarkable men in the history of American medicine, not because of anything that he personally accomplished — for a careful search of his works reveals not a single contribution to the healing art — but because he was wrong so often on such important matters and in such a violent manner. He was against everything new and he was against everyone who thought that anything new was good. He[16] quarreled with Channing and Simpson over anesthesia in childbirth, and he[4] berated McDowell when he removed ovarian cysts and thereby saved his patients' lives. Yet the world owes him some gratitude, for had he not assailed Holmes in such a bitter fashion the little doctor might never have returned to the attack.

What, in the history of puerperal fever, is the relation between Holmes and Semmelweis? As regards the transmission of ideas, there is no continuity whatever; for Semmelweis, who began his work three years after Holmes's essay, had never heard of the American. Semmelweis was appointed assistant professor in the *Frauenklinik* of the *Allgemeine Krankenhaus* in Vienna in 1846. Soon after assuming his duties he found that the mortality from puerperal fever among the patients delivered by the students was over

twice that among those under the care of the midwives. He also noted that few women died who had been delivered before they entered the hospital or who had entered in premature labor: the common factor was that none of these patients were examined. At about this time his friend Kolletscha died from a wound received in making an autopsy, and he found the same changes in Kolletscha's body as those that he had seen in women who had died of puerperal fever. He thereupon forbade students to come directly from the autopsy or dissecting rooms to deliver women in the clinic, and he ordered that everyone who examined a patient in labor should wash his hands with liquid chlorine or chlorinated lime. He was the first, therefore, to insist on disinfection of the hands at the time of labor; both Watson and Holmes had gone no farther than to advise it when leaving a case, so that one might avoid carrying contagion to the next patient. Immediately there was an improvement, and during the next year the death rate was cut in half.

In 1847 Semmelweis's results were published by Hebra,[4] his colleague, who followed with a second article in 1849.[4] It was not until 1861, however, that Semmelweis's book, *The Cause, Concept and Prophylaxis of Puerperal Fever*, appeared. Whatever advantage there is in priority, therefore, belongs to Holmes; although neither he nor Semmelweis, as is clearly shown by the authorities that the Bostonian quoted, was the first to advance the theory that childbed fever was contagious. As long before as 1694, Phillipe Peu,[17] of Paris, had said, "Putrid effluvia, exhaling from wounded men, brought on a fever which killed a great many childbed women who lay in the same hospital"; and he added, "Are not the putrid effluvia, arising from the lochial discharges in lying-in hospitals, capable of producing the same disease?" To Holmes, also, goes the credit for greater eloquence and vigor of expression; his essay is one of the literary masterpieces of medicine. But his premises were based entirely on the experiences of others; he had no personal knowledge of the disease. On the other hand, Semmelweis's conclusions sprang from his own clinical experiments; there was no need to summon other evidence; indeed, the fact that he knew nothing of the existing literature on puerperal fever adds, if anything, even more to his honors.

After Holmes and Semmelweis about forty years went by before epidemic puerperal fever was finally checked. In America the reactionary influence of Meigs and Hodge, and in Europe that of Semmelweis's opponents — led by Scanzoni,[4] of Prague — continued to cost the lives of countless women. There was more respect in those days for academic authority, there were fewer professors, and they were vastly more impressive; their *ex cathedra* statements had all the authenticity of gospel. Moreover, if the contagion was really carried from patient to patient, no one had yet demonstrated its nature or how it was conveyed, for the science of bacteriology did not then exist. In 1865, however, Lister,[18] inspired by Pasteur's discovery that micro-organisms caused fermentation and putrefaction, introduced the principle of antisepsis. To him obstetrics is as much indebted as is general surgery, for nine years later Bischoff,[19] of Basle, impressed by what he had seen in England, applied Lister's methods to women at delivery. Further steps toward the control of puerperal fever followed a logical sequence; in 1878 Koch[20] showed that bacteria exist on every unsterilized surface, and the next year Pasteur[21] discovered that the streptococcus caused the most virulent form of the disease. Using the antiseptic technic that followed such progress, Breisky,[22] of Prague, was able in 1882 to report that he had delivered eleven hundred women in succession without a single death — then an unheard of record.

Garrigues,[23] the first to introduce antiseptic obstetrics into America, reduced the death rate at the New York Maternity in 1885 from one in sixteen to one in seventy-two; and using the same methods his friend, Richardson,[24] of the Boston Lying-in Hospital, reported that during the following year no woman had died of puerperal fever, although for some time the mortality had been one in twenty. Simultaneously occurred the development of surgical asepsis, which was introduced by von Bergmann[25] in 1882. Finally, in 1894, came the publication of Tarnier's book, *De l'asepsie et de l'antisepsie en obstétrique*, which, except for a few minor points, described an aseptic regimen that is as sound today as it was fifty years ago.

As we bid farewell to puerperal fever and to Dr. Oliver Wendell Holmes, we are still puzzled by the question, Why did he abandon the practice of medicine? A man of his intellect and professional training, possessing his knowledge of humanity and his store of ready sympathy, seemed blessed with every essential quality that a good doctor requires. Perhaps he was too tenderhearted, for he was always distressed by suffering in any creature. Possibly, also, the fact that he wrote verses caused his patients to distrust him as a doctor. They forgot that the same god held sway over poetry and medicine; nor were they mindful of the fact that one might woo Euterpe and worship Aesculapius, and still be counted faithful to Apollo. Of course, Mr. Emerson, of Concord, was a poet, although at times he was a

little difficult to understand; so was dear Mr. Longfellow, of Cambridge, who wrote those lovely lines about the blacksmith and the chestnut tree; but, then, neither of them was a doctor. New Englanders in those days liked their medical men to be dignified; if they were portentous, pontifical, and prolix, so much the better. It may have been that Holmes's gay, untrameled spirit seemed out of place in the sickroom; it is also possible that the refined ribaldry that colored some of his utterances disquieted his patients. A man[26] who had referred in one of his lectures to the old anatomical plates of Spigelius as those "in which lovely ladies display their viscera with a coquettish grace, implying that it is rather a pleasure than otherwise to show the lace-like omentum and hold up their appendices epiploicae as if they were saying, 'These are our jewels,'" might well be too light-minded to be intrusted with the care of those who were seriously ill. Also, it was rumored that he had once defined woman as a "constipated biped with a pain in the back."

The definite break with practice came in 1857, when the *Atlantic Monthly* was founded and "The Autocrat of the Breakfast Table" began its appearance in serial form. From then on, aside from his lectures at the medical school, which continued until his retirement in 1882, he was primarily a man of letters. Perhaps, after all, the real answer lay in a remark he[27] made toward the end of his life about his medical friends: "I hope that they are not ashamed of me and do not reproach me for choosing the flowery path of very light literature rather than to chain myself to the heavy tasks of medical practice." And so it happened that Oliver Wendell Holmes is known to history as a poet, essayist, and philosopher, for there are few besides physicians who remember his part in casting out the evil spirit that in those days haunted the lying-in chamber; and so it happened also that although he is by no means Boston's most famous doctor, he is, without a doubt, the most famous Bostonian who ever practiced medicine.

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CARE OF THE VICTIMS OF THE COCOANUT GROVE FIRE AT THE MASSACHUSETTS GENERAL HOSPITAL*

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BOSTON

THE Massachusetts General Hospital has recently passed through a most instructive experience. On Saturday evening, November 28, 1942, the Cocoanut Grove, a Boston night club, caught on fire. As a result, 491 men and women lost their lives. During the two hours following the onset of the fire, 114 casualties were brought to the Massachusetts General Hospital. The majority of these were dead on arrival, many survived only a few minutes, but 39 lived and were treated.

The patients arrived in various states. A few came in taxis and walked into the hospital. Most were brought by ambulance on a stretcher. Some were carried in unconscious with no visible burns and died a few minutes after entry. A number were competent and lucid, many were maniacal. The surface burns of the early arrivals were limited to the head and hands; the clothing was but little damaged. Nearly all were coughing; some had labored breathing. Eyes, nostrils and mouth were often burned. Both the mucous membranes and burned surfaces of several patients were cherry red in color, indicating some degree of carbon monoxide poisoning. It was obvious almost at once that we were dealing with something more than the problem of burned skin; a severe impairment of respiration also existed. The possibility that there was pulmonary damage due to an explosion was considered until a few victims arrived who were able to give an account of the fire. It was then clear that no explosion had occurred but that suffocating fumes, in addition to flames and hot air, had been inhaled. An increasing proportion of the late arrivals were extensively burned and unconscious; their clothing was charred and soaking wet.

By this time the general pattern of the injuries sustained was established. The surface burns involved the head and hands, and in the women areas inadequately covered by clothing such as the neck, back, arms and legs. The burns were sooty and shaggy from ruptured blebs. In some the cherry-red color of the deeper burn tissue was vivid. Although burns of the upper respiratory passages were absent in many, signs of pulmonary damage were present in nearly all. There were no fractures in spite of the panic at the fire.

Had this not been a war year, the staff of the hospital would have been overwhelmed by so many victims. As it was, the hospital in many ways was prepared to receive them.

The hospital's preparedness was due to two of its war activities. First, since Pearl Harbor, the staff had been organized into teams to meet possible bombing disasters; drills had been held. Second, in two research projects, under the auspices of the Office of Scientific Research and Development, the problems of burns had been under investigation in the laboratory and clinic for nearly a year. Many of the staff had taken part in this research program. Newer modes of therapy had been appraised, and finally when a treatment for burns had been selected for use in a disaster, the staff was well aware of its use and purpose.

The importance of anticipating a disaster cannot be overemphasized. The casualties reached the hospital on an average of one every fifty seconds. It was necessary to dispose immediately of the dead in order to have room to care for the living. Four medical interns, working in pairs, were stationed at the admitting door to sort out the dead, who were taken along one corridor to a temporary morgue. The living were deposited, still on their stretchers, in the rooms and along the corridors of the Emergency Ward. Nurses and interns, usually in groups of four, were assigned to each casualty.

A room of the Emergency Ward had long before been set aside as a storeroom for emergency supplies. Wooden sawhorses were at hand to set the stretchers on. Sets for intravenous injection, sterile goods and ointment, enough to care for two hundred burned casualties, were immediately available.

The hour of the accident was fortunate in that the victims were arriving at the hospital at the time of the evening shift of nurses. Those nurses who were just coming off duty swarmed down to the Emergency Ward to aid those called from duty elsewhere in the hospital. The resident staff

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of eighty interns and residents were nearly all immediately available. From outside the hospital, medical students, nurses, nurses' aids and college students volunteered in numbers. Members of the nonresident visiting staff assigned to emergency duty, and technicians, were called by the telephone operators. Having at hand classified lists of the required personnel is imperative in such emergencies.

At first the emergency floor was cleared of all other patients. When it was suspected that the casualties might outnumber the bed capacity of the Emergency Ward, one entire floor of the surgical building accommodating forty patients was evacuated according to previous arrangement and set aside as an isolation ward. The former inhabitants of this floor were fitted into vacant places in other wards throughout the hospital. Subsequently a second floor in the surgical building was cleared, but was not needed.

The concentration of the casualties in one area, rather than their dispersal throughout the hospital, has merit. Not only was strict isolation against organisms of the respiratory tract immediately possible, but better supervision of the care of shock and anoxia was permitted. Emergency quarters hastily fitted up in corridors, garrets or cellars have not the facilities of running water, suction, piped oxygen and electric outlets so necessary for the care of the sick.

The treatment of most of the patients was begun while they were still on the stretchers in the Emergency Ward. After they had received an injection of morphine almost at the entrance, their burned surfaces were covered with sterile towels and intravenous fluids were started. The patients were undressed as they were transferred to beds and were wheeled in the beds from the Emergency Ward directly to the isolation floor. Here the burned surfaces were dressed. Members of the staff, doctors and nurses, were dispatched to this floor to carry on the treatment. The resuscitation squad assembled all the anesthesia machines in the hospital for oxygen therapy. By the time the last of the patients was transferred, the entire staff was working on the isolation floor. The patients were divided into groups of four and attended by members of the visiting staff, residents, interns, nurses and students. The responsible staff members of the burn and resuscitation squads acted as consultants for all the groups.

Within three hours of the time of the fire, all 39 patients were settled on the one floor and were receiving, when needed, oxygen for anoxia and plasma intravenously for shock, their surface burns having been protected by the permanent dressings.

The expeditious handling of these casualties was, of course, made possible by an adequate number of doctors and nurses being immediately available, and by the presence of a blood bank and sufficient supplies in the hospital. But the number of the personnel is of secondary importance to having a prearranged plan of action. Without forethought, both in regard to segregation of the patients and the type of therapy to be employed, confusion will reign.

Treatment

Prior to the Cocoanut Grove disaster, the members of the staff responsible for the care of burns had advocated a plan of treatment that emphasized primarily the prevention of the threatening complications of any severe burn, shock and infection. Shock can be treated, but it is wise not to allow it to get beyond the impending stage. Infection too can be conquered, but it is safer and more economic to take the pains to guard against it. In general, it had been planned to prevent shock by plasma administration, to guard against infection by strict isolation, aseptic technic and internal chemotherapy and to cover the surface burns with a bland ointment and pressure dressings and not to cleanse or débride them. This plan was adhered to but had to be altered to give precedence to treatment of the lung injuries. Respiratory complications such as the victims of this disaster exhibited had not been reckoned on, and they demanded immediate treatment.

Pulmonary Complications

As the hours passed after the disaster, the succession of pulmonary signs in the patients recalled those encountered in the soldiers of World War I poisoned by phosgene, and were also similar to those seen in the victims of the Cleveland Clinic disaster of 1929. It had been thought that this latter disaster was unique, for with the resulting change in the chemical composition of x-ray film it had not been expected that nitrous gas poisoning would again be encountered in civilian life.

The picture presented by the Cocoanut Grove patients on arrival was startling. The majority were either maniacal or unconscious. At first the mania was mistaken for hysteria aggravated by pain. Subsequently it was realized that in many it was due to anoxia, and that it was comparable to the restlessness and mania encountered in the anoxia of cardiac insufficiency.

Recognition of the part played by each of the three possible causes of such mania — pain, hysteria and anoxia — is important because the rational therapy for each differs.[1] Morphine is the treatment of choice for pain, but it may aggravate hysteria by its cerebral effect, and anoxia by its de-

pression of respiration. Morphine in large doses is a universal recommendation in the treatment of burns, and we employed it routinely, and without proper consideration, on the victims of this disaster. Each patient received ¼ gr. immediately on entry. If this did not have a quieting effect, often a second, and occasionally a third, dose was administered. It is probable that the initial dose did not materially depress respiration but did presumably bring comfort by relieving pain. An overdose, however, was certainly given in two or three cases. Respiration ceased in one patient; she was intubated and given artificial respiration with oxygen for a period of five hours, and survived.

Beecher[1] has pointed out that barbiturates given intravenously are better than morphine for the control of hysteria. None were used in these patients because of the large dose or doses of morphine that had already been given.

Obviously, the treatment for mania due to anoxia is the administration of oxygen. To those to whom it could be given it brought relief and quiet. Two patients, however, were so uncontrollable that it was impossible to restrain them to give the oxygen. To one of these, a strong young man, 4 cc. of paraldehyde was given intravenously and it had an immediate quieting effect. The other patient received paraldehyde intramuscularly with a similar effect. It was not given to any other patient, however, because of the fear, perhaps unwarranted, that it might add to the pulmonary irritation, since it is exhaled through the lungs.

Vomiting also added to the difficulty of oxygen administration. A number of the patients, including those who were unconscious, vomited during the early hours after arrival. Constant care had to be taken to prevent aspiration of vomitus. Oxygen therapy would have been carried out more easily in some patients could an intratracheal tube have been passed, but this would have required cocainization of the pharynx, which was not done because of the fear that vomitus would be aspirated following the loss of sensation.

The anoxia had two causes. The one which played the smaller role was carbon monoxide poisoning. Oxygen with 5 to 7 per cent carbon dioxide was given to the patients with signs of the poisoning. The major cause was pulmonary damage, which prevented the transfer of oxygen from air to blood. The signs of pulmonary damage multiplied hour by hour. Between the third and fifth hours after the accident, when edema of the skin burns became apparent, signs of pulmonary edema were also evident and anoxia was aggravated. Seven patients received oxygen therapy throughout this period, and suction of the upper respiratory tract was needed.

In spite of the edema of the airway and of the lungs, no patients died within the first twelve hours. The mania and vomiting decreased during this initial period and most of the patients subsided into either a coma or deep sleep.

Beginning twelve hours after the disaster, the clinical picture of the respiratory-tract complication assumed an ominous form. Obstruction of the bronchi and large airways menaced life. Seven patients died, all within a period of thirteen to sixty-two hours after the fire and all as a result of anoxia. No patient died after the third day.*

The lungs of the first patient to die had been gradually flooded with progressive edema. Oxygen under pressure was tried without benefit in such cases. The second patient to die, approximately twenty-four hours after the accident, developed acute respiratory failure, apparently due to edema of the larynx. Tracheotomy was done, but without avail. The third patient to die developed similar edema of the larynx, with sudden closure of the glottis. This man was a strapping young Army officer who had walked into the hospital with severe burns of the face, upper respiratory passages and hands. He had been so restless that he could not lie still but insisted on stamping around and waving his hands; such activity probably did not benefit his injured lungs. Within a few hours signs of pulmonary edema were obvious; tracheotomy was of no help. Following insertion of the tube into the trachea, he coughed up a long shred of mucous membrane, indicating severe damage deep in the bronchiolar tree.

The nature of the lung damage became clear after the post-mortem examination of 3 patients. In those patients severely burned about the face there was denudation of the mucous membrane of the nasal passages, mouth and pharynx. There was also obvious damage below the cords of the larynx, with shreds of mucous membrane and exudate heaped up at the upper end of the trachea. The midportion of the trachea was relatively uninvolved, but increased damage was again present at the carina.

The distal part of the bronchial tree showed shreds of edematous, denuded mucous membrane clinging in spots down into the small bronchioles. It was undoubtedly such shreds that several of the patients had coughed up at intervals. Where these shreds were held in situ, signs of occlusion of the bronchus were apparent. Patchy areas of atelectasis alternated with areas of emphysema. In those areas in which obstruction of the bronchus was complete, atelectasis had resulted; where incomplete, sufficient air had passed on inspiration to be trapped and to cause emphysema.

*One patient committed suicide on the forty-second day, a result directly attributable to the disaster.

The burns of the upper air passages were in part due to heat, but it is probable that toxic fumes accounted for some of the pulmonary irritation. One girl with no external visible burn developed moderately severe signs of irritation in the pulmonary tree. The other patients with minor burns of the face and none of the nasal passages, mouth or pharynx developed similar signs of plugging of the bronchioles.

In the patients who survived, this bronchiolar obstruction manifested itself in various ways. Some patients developed typical asthmatic breathing. At times this was relieved by both adrenalin and aminophyllin, and there was therefore undoubtedly an element of bronchiolar spasm, presumably as a result of the inflammation. From day to day, areas of atelectasis alternated with emphysema. Confirmation of the changing physical signs was obtained by frequent roentgenologic examinations. In one patient with no external burns, atelectasis of both lower lobes developed, accompanied by a vital capacity of only 800 cc. Continuous oxygen therapy was necessary for five days. On the ninth day, following postural drainage, she coughed up a large amount of mucocellular material, and the lungs then became filled with air. Throughout this period of atelectasis she was afebrile. There was undoubtedly a plugging of the larger bronchi. No signs of permanent damage have so far resulted.

Tracheotomy was resorted to but was of limited usefulness because the root of the respiratory evil lay distal to the larynx. It was used in 5 cases; only 2 of the patients survived. In one of these the burn of the pharynx was so severe that swallowing was interfered with. Feeding was carried on from the seventh through the sixteenth day by a tube inserted through the nose into the stomach.

The peak of respiratory difficulty passed on the third to the fourth day following the fire. Signs of moisture decreased and the injured air passages opened. Concern for the lives of these patients, however, was not over. Sepsis in the denuded pulmonary tree was still dreaded. Strict isolation of the patients was continued in the effort to exclude contaminating organisms from the respiratory tract, and no clinical or roentgenologic signs of pneumonia occurred. Virulent staphylococci were obtained on culture from the mucocellular material coughed out of the trachea, but no frank abscesses developed. By the end of two weeks the lungs of most of the patients had healed and our vigilance was relaxed. The unhealed lungs of 4 patients, however, kept them in the hospital two weeks longer.

Careful clinical and roentgenographic follow-up of all patients has shown a complete clearing of the lungs in all except 1 patient, who still had an unexpanded area of atelectasis in one lower lobe at one hundred and nineteen days. This patient had only a moderate degree of pulmonary damage and did not require tracheotomy. She was hospitalized for an extended length of time because of her severe surface burns. It is possible that the enforced inactivity was a contributing cause of the residual atelectasis.

Shock

The primary phase of burn shock is generally considered to be neurogenic. An imbalance of the autonomic nervous system results in a relaxation of the vascular tone and therefore in an increase of the capacity of the vascular bed and a drop in blood pressure. Pain is often a cause of this imbalance. Such primary shock was encountered in a few of the patients from the Cocoanut Grove. One man with moderate burns, who had been soaked by water and thoroughly chilled, had a systolic blood pressure of 80 on arrival. With rest, warmth and morphine his blood pressure promptly rose to normal. But primary shock was confused by anoxia, which tends to raise the blood pressure.

The secondary phase of burn shock comes on more gradually and is caused by the seepage of blood plasma out through the capillaries into the burned area, for the burn creates an abnormal permeability of the walls. Low blood pressure in this phase results from a decrease in the circulating plasma volume and an increase in the viscosity of the blood, the result of hemoconcentration.

The ideal treatment of secondary shock is the constant replacement of plasma as it is lost from the circulation. If this can be done by injection of plasma intravenously, the course of the patient through this phase, which generally lasts for thirty-six hours, will be smooth. If treatment is delayed until the shock state has already been reached, it is more difficult to return the patient's condition to normal. Nearly ideal conditions existed for the prevention of secondary shock in the patients from the Cocoanut Grove fire. The fire was but a mile from the hospital and all the patients were admitted within two hours of the onset. Adequate quantities of frozen human blood plasma were available in the hospital's own blood bank. The plasma had to be thawed, but to the late-comers was administered intravenously within five minutes of their arrival. Little or no true secondary shock was therefore encountered in any of the patients.

The damage to the parenchyma of the lung, however, created a dilemma so far as the administration of intravenous fluid was concerned. An integral part of the pathologic physiology of a thermal or chemical burn is an increase in capillary permeability in the area burned and its immediate environs. A protein-rich fluid, slightly more dilute than plasma, exudes into the extracellular spaces and out from the tissues where the surface is denuded. Such weeping occurs in burns of the lungs and bronchioles as well as those of the skin, and is responsible not only for the edema of the alveolar and bronchiolar walls but also for the fluid in the alveoli and lumens. The edema and fluid of course cut down the transfer of gases across the alveolar wall, and, when excessive, result in anoxia.

Edema in the burned area may be increased by the intravenous injection of saline or glucose, solutions of small molecules that are freely diffusible through the capillary wall. Such solutions increase blood volume, even transitorily, raise blood pressure, and thereby increase the outflow of the plasma filtrate from the capillaries. Restoration of normal blood concentration and volume by the administration of plasma itself probably also tends to increase the edema of the burned tissues, even though it maintains a normal circulation for the unburned portions of the body. It is possible that if a certain amount of hemoconcentration is allowed to exist, the plasma will seep out of the damaged capillary membrane less readily and edema will be decreased. The blood of the patients with the severe signs of pulmonary damage was allowed to concentrate to some degree. It is not clear whether this hemoconcentration was effective. It was not sufficient to disturb the metabolism of the patient as a whole, since normal kidney and liver functions were maintained.

Surface Burns

The recognition in the last half-century that burn shock is due to plasma loss rather than to absorption of toxin from the burn has created a debate concerning which should be treated first, the shock or the burn. Without proper treatment of shock, the life of the severely burned patient is in jeopardy. To maintain, however, as some still do, that nothing should be done about the burn wound itself until after the treatment of shock has been accomplished is to court trouble.

Neglect of the surface wound means increased bacterial contamination, and contamination means infection. The chief problem of burns is infection, and the most effective way to eliminate infection is to prevent it by covering the wound at the earliest possible opportunity. The importance of such preventive treatment cannot be overemphasized.

The treatment of the surface wound cannot be divorced from that of shock, for they are interrelated and must go on simultaneously. Infectious toxemia leads to shock, and shock leads to an increased growth of bacteria. Shock from an inadequate circulating blood volume leads to stagnation of blood, particularly in the peripheral tissues, and results in a decrease in oxygen tension. Some organisms, such as certain streptococci, are preferential anaerobes and multiply more rapidly under conditions of anoxia. From the point of view of prevention of infection, therefore, an adequate treatment of shock is imperative, just as is the prompt covering of a burn wound with a protective coating.

One of the drawbacks of the current methods advocated for the treatment of burn wounds is their complexity. It is common practice, both in civilian hospitals and in the armed forces, to débride and cleanse such wounds. Débridement consists of opening all unruptured blebs and cutting away the dead epidermis overlying these blebs and those that have already ruptured. This leaves the dermis exposed, a surface vulnerable to infective organisms. Therefore, such exposed surfaces are gently scrubbed with soap and water or a detergent, and finally rinsed with saline solution. Since such maneuvers may be exquisitely painful, even when deftly performed, anesthesia is often considered necessary. Such débridement and cleansing are conducive to further shock, particularly when accompanied by general anesthesia, and are postponed until the treatment of shock is advanced. Anything that aggravates shock is obviously contraindicated. Such débridement was a necessary preliminary step for the treatment with tannic acid. It has been carried over into other forms of treatment without critical evaluation.

Another relic of the tannic acid ritual, which adds to the complexity of surface treatment, is the two-stage treatment of the burn surface, first-aid and definitive. Débridement and hourly spraying of tannic acid cannot be carried out under first-aid conditions but have to be reserved until operating-room facilities are available. This increased handling is also conducive to shock and is likewise contraindicated.

For several months prior to the disaster the rationale of reducing the treatment of the burn wound to the simplest terms had been explored in the clinic and laboratory of this hospital. Such a simple surface treatment had been tried out, but received its first mass trial on the victims of this fire.

The extent of the surface burn of these patients varied from patches of the mildest first-degree burns to extensive deep burns. The burns of the typical pattern were not extensive but were frequently deep. One third of the patients had burns that were more widespread than the general pattern, and these were deep. Many of the burned areas were grossly clean, others covered with dirt and soot. A number of the patients had been crawling on the floor at the fire or were piled in with the dead. The burns of several were exposed to feces, and many of the patients, dead and living, were incontinent. The eye burns were limited to the lower half of each cornea.

The treatment was carried out as follows: The attendant personnel was carefully masked to prevent the spread of organisms from the upper respiratory tract.* Immediately all exposed burned surfaces were covered with sterile towels. When the patients were undressed and transferred from stretchers to beds, burns of the body were covered by laying sterile sheets on the bed and enfolding the patient in them. On the isolation floor these sterile drapes were removed and the surfaces covered with boric-ointment gauze. The gauze, impregnated with liberal amounts of the ointment and autoclaved, was put on by medical students and interns who wore, in addition to masks, sterile gloves but unsterile gowns. *No débridement and no cleansing of any surface were done.*† Dry sterile gauze was laid over the ointment gauze and pressure bandages were applied to all wounds of the face, scalp and extremities. Sulfathiazole, 5 per cent, in petrolatum was placed in the burned eyes, the lids closed, and the eyes incorporated in the pressure dressings of the face. Only the nostrils and mouth were left uncovered.

As a final but integral part of the surface treatment, 2 gm. of sodium sulfadiazine was injected intravenously into each patient. This was accomplished readily by a technician who injected it into the rubber tubing of the intravenous set, which was already functioning. Subsequent doses of sodium sulfadiazine were given intravenously, or by mouth if the patient could tolerate it.

Such a treatment of surface burns is unorthodox in modern surgery, when one is accustomed to neat wounds. When it was first suggested a year ago that we should not débride a burn wound, this was objected to by those concerned with infection. They believed that all blebs must be opened because otherwise the bacteria harbored in gland crypts would burrow up from the base of the vesicle to infect a fluid that was a fertile anaerobic culture medium. This objection seemed unreasonable on two counts. In the first place, skin has been shown to have the ability to destroy pathogenic organisms on its surface,[2-4] and it is probable that the gland crypts have the same ability. The only organisms to survive on skin are its natural habitants and they are nonpathogenic. These organisms, principally *Staphylococcus albus*, should not lead to an infectious process. In the second place, in the early hours of the burn at least, bleb fluid is in open communication with the extracellular spaces. In 1932, Field, Drinker and White[5] showed that there is a greatly increased flow of lymph from the area of an experimental burn, suggesting that there is a turnover of fluid in a burn wound. Since the fluid originates in the capillaries, it has the oxygen tension of the blood, and relatively aerobic conditions may well exist in a burn wound until the intercellular spaces become plugged with fibrin. An observation of comparable significance to the increased lymph flow in the dog has often been made in the human being: a débrided burn wound weeps freely, and the protein-rich fluid from the capillaries brings with it a supply of oxygen to the injured tissue.

It remained to be shown in patients with burns that blebs left undébrided did not become infected. Prior to the Cocoanut Grove disaster, the unruptured blebs of 26 patients, some with several blebs, had been protected from one to fourteen days, after which time the fluid was withdrawn from these vesicles under sterile conditions and cultured. The fluid from the majority of blebs was sterile; from a few the nonpathogenic saprophytic organisms of the skin were recovered, and in only one was a virulent organism, a streptococcus, cultured. In this case alone was the bleb grossly infected. In all the others, healing occurred promptly beneath the unruptured blebs and there was no evidence that the bleb fluid retarded healing.

Again, we were unorthodox and did not cleanse the wounds because we doubt that any cleansing other than vigorous scrubbing materially reduces the number of organisms on the wound. Such scrubbing of delicate, unprotected tissue would presumably result in more extensive injury and as much harm would be done as benefit derived.

A simple bland ointment was applied to the burn surface because, so far as has been determined, such an ointment does not injure the viable remnants of epithelium. Substances such as tannic acid or the triple dyes commonly recommended for the treatment of burns have recently been shown to retard the regeneration of epithelium.[6] It is possible that plain petrolatum would be as good as or better than petrolatum with boric

*Masking of the patients was impossible because of the respiratory complications.

†The face and left hand of 1 patient were partially débrided and cleansed with soap and water before the general order was understood.

acid. Boric acid, however, may prevent the multiplication of pyocyaneus organisms; pyocyaneus was conspicuous by its absence in these patients. On the other hand, too rapid absorption of boric acid might result in poisoning. Boron was recovered in the urine of these patients, but no poisoning attributable to its presence was determined.

No substance has been proved to promote epithelial regeneration. It is believed by many that toxins are formed in burn wounds, but their presence has not been substantiated and surface agents designed to eliminate them are, therefore, of questionable value.

A sulfonamide was not incorporated in the ointment, not because we believe that it has an injurious effect on the epithelium but because absorption through the burn wound occurs and may be rapid and irregular and give rise to sulfonamide poisoning.[7, 8] The sulfonamide administered internally permeates through the burn wound since it is freely diffusible out of the capillary wall. It is possible to control the level of sulfonamide in the body fluids by internal administration and thus prevent poisoning. At this hospital, sulfonamide levels have been demonstrated in bleb fluid equal to those in the blood plasma. Such observations were also made in the patients of the Cocoanut Grove fire. We are not certain, however, if sulfonamide therapy is withheld for more than forty-eight hours, that an adequate bacteriostatic level in the burn wound would be reached, since the cell spaces eventually become plugged with fibrin.

The original dressings were not changed on any of the patients until the fifth to tenth day, when a boric ointment dressing was reapplied. Full precautions were maintained in a temporary operating room set up on the isolation floor. Later, in preparation for grafting, saline or boric acid solution dressings were used to help liquefy the dry slough of the deep burns.

The main advantage of this surface treatment lies in its simplicity. In a civilian disaster, as in warfare, there is inevitably a disproportion between the number of casualties and the number of trained personnel. This was true in this disaster. The treatment was applied by relatively untrained workers, the skilled personnel of this hospital being free to care for the pressing problem of anoxia and shock. The activities required to apply the dressings were so unobtrusive that the administration of plasma and oxygen was not interfered with. The dressing relieved pain promptly, and all manipulations that tend to increase shock were cut to a minimum. Since the dressing does not interfere with the life-saving treatments and does not increase shock, it could be applied immediately and therefore contamination and subsequent infection were reduced. It is so simple that hospital facilities, such as an operating room, are not required. The treatment is both first aid and definitive. Once applied, the dressings should not be touched for a week to ten days.

Because of its simplicity, this method of treatment has administrative advantages. It requires no complicated apparatus and nothing that is not used in other types of wounds. Special solutions, spray guns and heaters, all requiring extra space for storage or transportation, are not needed.

The results of this simplified treatment of the burned surface were gratifying. The second-degree or incomplete-thickness burns of the skin healed promptly without evidence of infection. The 17 patients having such burns had all been discharged by the end of two weeks. The only patients remaining in the hospital for a longer period were the 10 with deep or complete-thickness burns of the skin, the 1 with severe damage to the central nervous system from anoxia and the 4 held on account of residual signs of the pulmonary complications.

Early grafting of the deep burns was possible. The sloughing dead skin contained numerous organisms and there were occasional small abscesses beneath the necrotic tissue, but the wounds were remarkable because of the absence of invasive infection. The margins of the skin next to the granulating areas were not ulcerated or inflamed. There was no evidence of cellulitis, lymphangitis or lymphadenitis.

This absence of active infection is to be attributed to the chemotherapeutic program, not to any cleanliness of the burns on arrival. None of the burns were cultured before they were dressed originally because of the exigencies of the moment, so that the actual degree of contamination is not known. The first cultures were taken at the time of the first dressings. Cultures were also taken at subsequent dressings and the course of the bacterial flora was studied until healing occurred. The bleb fluid of unruptured blebs was cultured in many patients and found, as expected, to be sterile. Pathogenic staphylococci were found in almost all open wounds in the first and subsequent cultures, whereas streptococci were almost completely absent. In any similar group of burn patients the latter organism would have been present in the majority as a result of contamination from the respiratory tract. It is fair to believe that the sulfadiazine was responsible for the incongruity of incidence of these two types of bacteria.*

*Although penicillin was administered to 13 patients, it was given in what is now known to have been too small a dose, and it was not started until the sixth day. No decision can be reached regarding its effect, for it is possible that the observed results are attributable to the sulfadiazine alone.

The burns of 9 of the patients were grafted. The extent of the surfaces grafted varied from 1 to 28 per cent of the body. The first graft was done on the twenty-third day, the last on the one hundred and twenty-fourth day. The initial graft on 2 patients did not survive, presumably because of the bacterial flora present in the granulations. The subsequent grafts on these patients took, as did all those on the other patients. The first of the grafted patients was discharged on the fortieth day, the next to the last on the one hundred and third day. All patients were discharged healed except for the last one, who was discharged on the one hundred and forty-third day. She was the most extensively burned patient to survive. On discharge there were still a few small granulating points in the grafted areas.

Many of the patients with deep burns of the hands have required physical therapy to restore function.

Clinical proof of the absence of invasive infection in the majority of the granulating areas of these patients is illustrated by the achievement in 1 patient. The skin on the dorsum of one hand was so severely burned that when the slough was cleared away the extensor tendons of two fingers lay exposed. A split graft to this area, had it been able to obtain an adequate blood supply, would have resulted in adherent tendons. Instead, the hand was implanted into the abdominal wall for two weeks. The graft of skin and subcutaneous fat took without incident and the tendons were saved. While the hand was in the abdominal wall there were minimal signs of inflammation. Had there been an active infection in the hand wound, the graft would not have taken and an abscess would have developed in the abdominal wall.

The absence of invasive infection not only permitted early successful grafting but also prevented any extension of the areas requiring grafting. Several deeply burned areas, at first considered to be complete-thickness burns, eventually epithelialized from numerous small islands of the rete. Had there been deep infection such islands would not have survived, and grafting would have been necessary.

Keloid overgrowth has formed in some of the deeply burned, ungrafted areas. It is too early to tell what will have to be done to them. Excision with grafting may be necessary, in which case it would have been wiser to have grafted the areas in the first place.

Pressure dressings have recently been advocated to reduce edema and promote healing. Although all the burns of the extremities, face and scalp were treated in this way, we do not think that we can commit ourselves concerning its worth. On the extremities, the pressure undoubtedly did reduce the edema, the fluid being expressed upward above the dressings. On the face the fluid was expressed posteriorly to the scalp and then downward into the deep tissues of the neck and over the chest and shoulders. The dressings formed effective splints and, by decreasing the amount of lymph, may have reduced the spread of infection.

Ligation of veins for thrombophlebitis was required in 5 of the patients. The lesion was first suspected in 2 patients by the appearance in chest roentgenograms of areas suggesting pulmonary infarcts. In the other patients, local inflammation was the presenting sign. The cause of the phlebitis in one case was the use of an ankle vein for a constant intravenous injection; the superficial saphenous vein was ligated on the ninth day after the fire. In the other patients there was probably more than one cause, prolonged bed rest with immobility being the most important factor. In two cases, a pressure bandage applied to wounds of the thighs and not started at the ankle was probably a contributing factor. The thrombophlebitis bore no relation to the chemotherapy or the multiple femoral-vein punctures that a number of the patients required for blood studies, administration of plasma and whole-blood transfusions.

Social and Psychiatric Aspects

A civilian disaster may induce social or psychiatric difficulties among either the survivors or the relatives of the dead. States of confusion, bewilderment and grief, requiring a psychiatrist's help, were encountered by the social-service workers among the relatives who came to the hospital seeking missing ones. Several of the injured had lost a husband, wife or close friends. One had lost a son and husband, and another, both her parents. Some of these patients made a good adjustment to their grief, others a poor one. All were aided by consultations with the psychiatrist. One patient who had lost his wife voluntarily returned to the hospital two weeks after his discharge for psychiatric care and eventually committed suicide. Although he had previously been an unstable person, his death is directly attributable to the disaster. The wisdom of the Social Service Department in its trained handling of relatives, and its prompt recognition of psychiatric disturbances, were noteworthy.

Metabolic Studies

The numerous studies on blood, urine and feces that were made in order to guide the course of the therapy have yielded considerable information. No patient developed impairment of kidney

function, and this in spite of the fact that hemoglobinuria was present in 9 patients, being massive in 5. Albuminuria was encountered but left no residual.

Metabolic observations were also undertaken. A study of the nitrogen balance was made in 29 patients. In 23 of these the 17-ketosteroid excretion was followed throughout the hospital stay and, in some, longer. In 8 patients the potassium balance was studied. When an adequate nitrogen and caloric intake was possible, nitrogen balance was maintained. The excretion of nitrogen by the kidney was not influenced by the extent of the burn but was influenced by the sex of the patient. During the initial days when there was an inadequate caloric and nitrogen intake, the nitrogen balance was negative but there was not a comparable negative potassium balance. This indicates that the source of the nitrogen was not cellular protein.

The nitrogen deficit was less than anticipated in view of recent reports.[9-11] It is possible that this relatively low excretion of nitrogen may be attributed to the absence of infection in the burn wounds, since it is well known that infections are associated with a rapid excretion of nitrogen from the body.

The excretion of 17-ketosteroids in the urine rose above normal during the first five to seven days and then abruptly descended to a subnormal level; this level was maintained until convalescence was complete. The steroid excretion did not correlate with the changes observed in nitrogen balance. Such a relation has been reported elsewhere, and it is claimed that the adrenocortical hormone is responsible for the excretion of nitrogen by burned patients.[11]

Comment

Many lessons can be learned from the experience with the victims of the Cocoanut Grove fire. Some were known before but their significance has been re-emphasized. The necessity for a prearranged plan of organization, administration and therapy for a large number of disaster victims is obvious.

Three important things in regard to burn therapy were learned. First, lung damage, presumably from both heat and irritating gases, may be an added complication and should be anticipated in any mode of therapy to be used on patients of a civilian fire. Unless one is aware of such a possibility, the meager initial signs in the lungs may be overlooked, and pulmonary edema and sudden death will result. The second thing learned, a corollary of the first, is that the administration of morphine in large doses should not be given to burned patients when pulmonary complications exist. If mania is present, anoxia should be excluded as a cause before giving a respiratory depressant, although in burned patients covered with dirt and soot, anoxia may be hard to recognize. Thirdly, the treatment of the burned surface can be simplified, without compromising the care of the wound, by dispensing with débridement and cleansing, provided that adequate chemotherapy is administered. Also, a complicated surface agent is not necessary to obtain gratifying results.

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Discussion

Dr. C. R. Mullins, Concord: I should like to know a few more details of the chemotherapy. What blood levels did you maintain, how often did you have to give the sulfadiazine, and what were the doses?

Dr. W. J. P. Dye, Wolfeboro: I should like to ask Dr. Cope a question concerning his method of administration of intravenous sulfadiazine. I had been under the impression that it should not be given at the time of or in conjunction with intravenous saline. Dr. Cope stated that this was administered through the tube in which saline and plasma were being administered.

Dr. R. W. Robinson, Laconia: For the last two years, in the group where I am practicing, we have been using a 15 per cent phenol and vaseline ointment, without débridement, when dealing with burns, and I am confident that I have never seen any débridement of a surface burn that affords such complete relief or in which the burns heal so quickly and so readily with as little scarring. This was brought to our attention through a Dr. Miller, who has an extensive practice in a West Virginia hospital in the coal-mining district; he has been using this ointment for years. I was a bit hesitant at first because of the fear of phenol absorption and poison. Of course, as an antiseptic phenol is excellent. Furthermore, epithelialization of these wounds takes place underneath the ointment, seemingly at an accelerated rate.

In the beginning we used it in only a few cases, but we came to employ it more and more extensively. Our pathologist covered the wounds of laboratory animals with it, in an attempt to determine whether there was

any evidence of absorpion of phenol. There was no such evidence whatsoever. In addition, patients who had been treated with this ointment and who had extensive burns, such as a whole leg or a leg and an arm or the abdomen, showed no evidence of phenol absorption, according to laboratory tests on the urine.

In other words, we have used no chemotherapy with this ointment, because we have had no infection whatsoever in the burned areas since using it.

DR. A. L. MACMILLAN, JR., Concord: I should like to ask Dr. Cope to say a word or two about burns of the cornea.

DR. COPE (closing): Perhaps I can answer the first two questions together, since they deal with chemotherapy.

All the patients had intravenous tubes in place, as I said. Each patient received 2 gm. of sodium sulfadiazine, injected intravenously by a technician. This was a simple procedure. Sodium sulfadiazine is a soluble salt; it is not like sulfadiazine alone. We have encountered no trouble from its use. It has been adopted as a routine procedure by the doctor who is in charge of surgical infections in our hospital.

The subsequent administration of sulfadiazine was by the continued intravenous route in those patients who could not swallow, and by mouth in those who could swallow. Each patient received an additional 6 gm. of sulfadiazine in the first twenty-four hours, and 4 gm. on the subsequent days, until it was omitted.

A few cases showed red cells in the urine. The blood levels, however, were carefully watched, and ranged between 4 and 10 mg. from the second day on. One patient reached 12 mg. on a single day. In the first twenty-four hours, the dose was not sufficient in 3 patients. Three patients developed sensitivity. One of them developed a drug fever and rash at the end of the second week, and the sulfadiazine was omitted.

Again, I want to emphasize that we consider this a successful therapeutic program. There are many things that enter into such a procedure against infection. First, the burn wound was covered early and protected immediately, thus reducing contamination. Secondly, the wound was not disturbed, reliance being placed on internal chemotherapy. Sulfadiazine was found each time that fluid from an exposed bleb was removed and analyzed, and its concentration was equivalent to that in the blood during the first ten days. However, if internal chemotherapy had been postponed until after forty-eight hours, I doubt that adequate levels in the bleb fluid would have been reached. By forty-eight hours, the intercellular spaces of the burned wound became blocked with fibrin, and passage of substances from the blood into the burned area and the bleb fluid was slowed.

DR. DYE: What were the contraindications?

DR. COPE: We did not believe that there were any.

As to Dr. Robinson's 15 per cent phenol in vaseline ointment, my objection to this is mainly that such a concentration of phenol undoubtedly damages otherwise viable epithelium, just as tannic acid does, or the triple dyes. A concentration of such a substance as phenol or the triple dyes that is sufficient to prevent bacterial growth is injurious to tissue. The sulfonamides and penicillin have the distinct advantage of being noninjurious to epithelium. They do not impede or retard epithelial regeneration in concentrations adequate for bacterial stasis. In that way, they are a godsend for the treatment of wounds.

As to the absorption of phenol, one might get a false picture by measuring or looking for it in the urine, because phenol is a protein precipitant and is rapidly fixed in the tissues. If any of it should get into the lymphatics, it would almost certainly be combined with protein; in this form, it might not be excreted by the kidneys promptly, yet could cause damage elsewhere. However, I should exclude phenol principally on the fact that it causes further injury. This is equally true for the triple dyes and tannic acid, if the concentration is high enough to keep down the infection. In deep burns that are incomplete when one puts on something that injures the epithelium, a full-thickness burn is created and the injury is increased, even to the point of requiring grafting, which prolongs hospitalization. That is the reason why the use of such substances is, in my opinion, to be excluded.

About the burns of the cornea, there were 12 patients out of the 39 who had bilateral ulceration of the lower half of the cornea. At the Boston City Hospital, patients with burns of the eyes had the identical lesions. The treatment by our ophthalmologist was 5 per cent sulfadiazine ointment in petrolatum, put under the lid; the eyes were then closed, and a pressure dressing applied. The corneas in all the cases healed without scarring.

THE GASTROINTESTINAL SYMPTOMS OF HEART DISEASE*

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MANY persons with heart disease first consult a gastroenterologist because of symptoms of indigestion. On the other hand, an equal number consult a cardiologist because of symptoms that suggest heart disease but in reality arise from the gastrointestinal tract. Not infrequently the physician himself finds it difficult to decide on the source of the symptoms.

CONDITIONS ENCOUNTERED

Congenital Abnormalities

Gastrointestinal symptoms may arise from congenital abnormalities of the cardiovascular system. A right-sided aorta, a rare anomaly, causes difficulty in swallowing by pressure on the esophagus; it is easily demonstrated by fluoroscopy, although there is little reason to suspect it before this is done. Congenital heart disease in infants is not infrequently associated with malnutrition, because of frequent cough and vomiting, which often result in a difficult feeding problem. In later life the gastrointestinal symptoms of congenital heart disease are much the same as in other forms of heart disease when myocardial insufficiency has developed.

Aneurysm and Mitral Stenosis

Disease of the great vessels, particularly aneurysm, may rarely cause dysphagia. Rheumatic heart disease with mitral stenosis and auricular dilatation may at times produce considerable pressure and displacement of the esophagus, thereby causing noticeable difficulty in swallowing solid food.

Angina Pectoris

The pain of angina pectoris is characteristically substernal, as is the pain of coronary thrombosis; however, both show considerable variation in distribution. Radiation of pain into the jaw and teeth or into the throat is by no means rare. The spread of pain into the epigastrium, below the xiphoid, is comparatively rare but sometimes occurs in patients with a pendulous abdomen. Patients are apt to consider low substernal pain, however, as arising from the stomach. When the pain of coronary thrombosis is associated with gas, nausea or vomiting, or any combination of the three, as is frequently the case, suspicion is naturally directed to the gastrointestinal tract.

The diagnosis of angina pectoris depends entirely on a carefully taken and evaluated history. Generally speaking, the patient notices the following: The pain or discomfort is brought on by effort or emotion. Secondly, the pain may be less severe or absent if he walks when his stomach is empty or aggravated if he walks after eating a heavy meal. The patient thus relates the pain to his digestive tract. The pain may be less severe if the weather is warm or greater if it is cold. The pain may be further aggravated when walking if the patient is emotionally upset. The worst possible combination for the anginal patient, therefore, is cold weather, difficult footing, a full stomach and an upset mind. Thirdly, the patient even while walking often notes a relief of pain by the simple act of belching. This phenomenon is not easily explained, and it is even more difficult to make clear to the patient that his pain originates in the heart and not in the stomach.

Nocturnal pain and gastric distress due to coronary disease may result from overeating. However, it may be a serious sign, often heralding a major coronary infarction.

Nausea is a common accompaniment of coronary occlusion, but I have seen it only once as the major symptom of angina resulting from effort. It appeared and disappeared with substernal and epigastric distress and was readily quieted by nitroglycerin or cessation of effort.

Nausea and vomiting in an elderly patient may be the only sign of coronary thrombosis of the silent type. Much may depend on the patient's interpretation of his low sternal or epigastric discomfort and on whether he calls such discomfort pain. However, one can frequently obtain the usual history of distress by careful questioning. Care must be taken in evaluating nausea and vomiting when other gastrointestinal lesions are present, for example, carcinoma of the stomach, ulcer and gallstones. In these cases, electrocardiograms, especially serial ones, are of decided value. Differentiation of ruptured ulcer or other acute upper abdominal diseases, such as acute pancreatitis, gall-bladder colic or upper intestinal obstruction, is thus made possible. Difficulty arises, however, in cases in which electrocardiographic changes are absent, and a decision concerning exploration must be made. A careful appraisal of the patient's history is most helpful. In spite of all the evidence that can be collected, a decision

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may be impossible and surgery may be undertaken only to reveal a normal abdomen. Fortunately, these cases are infrequent.

Congestive Heart Failure

Acute cardiac decompensation with sudden distention of the liver may also lead one to suspect an acute upper abdominal emergency. However, other clinical signs pointing to a satisfactory diagnosis should be present. The presence of valvular disease, cardiac enlargement, dyspnea and even cyanosis should be striking enough so that an acutely engorged liver is not confused with other upper abdominal lesions.

With insidious congestive failure either from coronary disease or from hypertensive or valvular disease, the gastrointestinal symptoms are less acute and have no constant characteristics. Loss of appetite, gas and vague abdominal distress may result. In these cases, orthopnea may not be so marked, and liver enlargement may be of only moderate degree. Tenderness on palpation of the midepigastrium with resistance there and dullness over Traube's semilunar space should always make one suspect enlargement of the liver. When this is due to congestive failure, the neck veins are almost always engorged, particularly when the patient is in a recumbent position but also discernible when he is upright. This type of failure is often unrecognized in coronary disease. There may be an antecedent history of substernal pain. The physician should be alert to tachycardia and gallop rhythm or a ticktack quality of the heart sounds. A cough may lead one to suspect the heart; in fact, an acute bronchitis with severe coughing may precipitate heart failure. The cough then persists as a symptom of pulmonary congestion, and with digestive disturbances the true etiology of the symptoms remains obscure.

When congestive failure reaches the point where ascites is present, bowel function becomes poor, resulting in increasing gaseous distention, anorexia and indigestion.

This whole train of symptoms is also present in chronic passive congestion of the liver associated with ascites caused by chronic valvular disease or constricting pericarditis. Upper abdominal pain after eating may be so severe as to lead one to suspect gall-bladder disease. If congestion can be relieved, the pain may well disappear. In some cases of long standing, hepatic cirrhosis develops, often associated with some degree of jaundice. These patients are difficult to handle and may never be free of gastrointestinal symptoms. Boyer and White[1] have called attention to pain in the right upper quadrant on exertion associated with right-sided heart failure. In my experience, this has not been encountered commonly, but like many symptoms often unnoticed it may occur more frequently than one suspects.

Embolism

Embolism is one of the most frequently undiagnosed causes of acute gastrointestinal symptoms, and may be pulmonary, renal, splenic, mesenteric or even cerebral. Perhaps the most frequent cause of embolism is rheumatic mitral stenosis, particularly when auricular fibrillation is present. Auricular fibrillation without valvular disease occasionally results in embolism. The mural thrombus of coronary infarction producing embolism may further complicate the gastrointestinal symptoms of this disease. Subacute bacterial endocarditis is another condition in which embolism may occur, as well as the whole train of digestive symptoms that accompany a severe infection. The pain may simulate gall-bladder or renal colic or appendicitis. The sudden onset of severe pain in a known cardiac patient is the keystone of diagnosis, and such a history should make the surgeon suspect the heart. A patient with mitral stenosis and auricular fibrillation should be warned that if such sudden attacks occur, he should immediately consult a physician and suggest the possible cause, thus averting serious and unnecessary operations. Should a diagnosis of mesenteric embolus be made, sufficient time (usually twenty-four to forty-eight hours) should elapse to permit demarcation to take place so that resection will be adequate.

Diaphragmatic Hernia

Certain gastrointestinal lesions frequently cause symptoms that simulate heart disease. Perhaps foremost among these lesions is diaphragmatic or hiatus hernia. This may produce substernal pain extending upward and may be associated with dyspnea, cyanosis and even shock, thus simulating acute coronary infarction. At times morphine must be given to relieve pain. The attacks may be frequent or infrequent. I remember a patient who was supposed to have had two coronary attacks, during which it was difficult or impossible to swallow food or liquid. This gave the clue. Although previous gastrointestinal studies had been done, the hernia was not found until the third examination.

Some of the most incredible complaints from an organic cause seem at first to have no such basis. All physicians tend to ridicule such complaints and classify the patient as being neurotic or as having hallucinations. However, one such case may make one more attentive to bizarre stories. A woman over fifty years of age complained of a loud swishing noise occurring occasionally

after meals and synchronous with her heart beat.[2] It could be heard ten or fifteen feet away. Examination and fluoroscopy of the heart and chest were negative. Not until some barium was swallowed was a large hiatus hernia discovered. When water and a liberal supply of soda were given, the heart beating against the entrapped portion of the stomach in the chest produced the churning motion of water and gas, thus causing the sound.

Anemia

Diaphragmatic hernia may cause symptoms of coronary disease in another way. Bleeding is not infrequent and marked anemia, with resultant anginal pain and considerable dyspnea on effort, may result. The bleeding may be difficult to control, but once the anemia is gone, the anginal symptoms usually disappear.

Cancer, particularly of the proximal colon, being often the unsuspected cause of anemia, may be another such instigator of cardiac symptoms. Indeed, anemia from any cause may play the same role. Severe congestive failure from anemia alone has been noted on several occasions.

Acute Dilatation of Stomach

Acute dilatation of the stomach after surgical operations may produce so much distress, along with tachycardia, dyspnea and even cyanosis, as to lead one to suspect some pulmonary or cardiac complication. Wise is the man who does not forget the usefulness of the stomach tube. The reverse may also be true,—that is, cardiac failure may cause dilatation of the stomach,—and although this is not common, the stomach tube should be used whenever it is suspected.

Disturbances of Rhythm

Functional cardiac mechanisms, such as paroxysmal auricular fibrillation, flutter and paroxysmal tachycardia, are frequently associated with indigestion. Premature beats, too, are at times a complaint. The latter are likely to be more noticeable during indigestion and are really unrelated to any gastrointestinal disturbance. Every physician has seen patients who had premature beats during examination but who were not conscious of them. Some patients think that paroxysmal auricular fibrillation is precipitated by gastrointestinal upsets, but when it comes to anything specific in the way of quantity or type of food, evidence is lacking. The only precipitating cause that I have encountered and that appears to have some relation is an alcoholic bout, particularly in persons who do not usually drink to excess. The withdrawal of tobacco, coffee and other suspected factors fails to prevent recurrence of these cardiac disturbances.

Many physicians apparently believe there is some relation between gall-bladder disease and functional heart disturbances or coronary disease. The relation between gall-bladder disease and functional heart disturbances is not impressive when viewed critically. However, gall-bladder disease and coronary disease are undoubtedly frequently associated, and the disturbance created by gallstones may aggravate the onset of anginal pain. Possibly the two diseases have a common cause, namely an abnormal deposition of cholesterol.

No precipitating causes of paroxysmal tachycardia have been discovered. In this disorder, as well as in paroxysmal auricular fibrillation or flutter, a sensation of fullness, gas or even nausea may occur during a prolonged attack. Early in life these attacks are well borne unless severe heart damage is present, but in the later years of life such tachycardias may, because of diminishing coronary circulation, produce substernal or high epigastric pain indistinguishable from anginal pain or that of coronary occlusion. Paroxysmal fibrillation is more likely to precipitate failure, not immediately but later, giving rise to the symptoms described under failure.

Symptoms from Treatment

Gastrointestinal symptoms due to treatment may be overlooked or unrecognized. The most important symptoms are those following the use of digitalis. Some patients cannot tolerate digitalis in doses below those that usually produce saturation, namely 1 to 2 gr., or 1 cat unit, per ten pounds of body weight. It is unwise to doubt the patient's statement that such is true. The claims made for various commercial preparations in this regard have little clinical proof. One should not inject intravenously or intramuscularly more than a quarter or a half of a theoretically digitalizing dose unless one is sure that no intolerance exists. Even if no intolerance exists, a calculated dose based on 1 cat unit per ten pounds of body weight should never be given intravenously in one injection but in divided doses not oftener than every three hours. The oral use of Urginin or the oral or the intravenous use of Cedalanid (lanatoside C) may in some cases be less liable to produce toxic symptoms.

Intolerance to such drugs as theobromine and ammonium chloride or even mercurials may prevent their use. Theophylline derivatives, especially theophylline sodium salicylate, seem to produce much less gastric irritation. Attention should be directed to the train of symptoms that may occur from too rapid or too great loss of fluid following diuretics. Two effects may result,

the first from concentration of digitalis bodies in the system, the second from excessive loss of sodium as the result of diuresis. Sudden and obscure deaths may have resulted from the above.

Treatment

In the treatment of these gastrointestinal symptoms, as well as of the heart itself, certain procedures should be avoided. Drastic purges are condemned, for although gas and distention may be relieved temporarily, they will recur. It is far better to avoid laxatives and saline cathartics and to rely on a low-roughage diet assisted by enemas of saline solution. A milk diet is suitable for some patients, but generally speaking it fails to keep the patient in a contented frame of mind. In my experience, a soft-solid diet with food that appeals has the advantage. In edema, the diet should have a low salt content, but not when it produces complete anorexia. The same is true for all drugs used. An attempt should be made to keep the patient happy, and to many patients this means a desire for and enjoyment of food. Thirst is often great. A daily fluid intake of 1500 cc. is handled well in most cases; less than this is rarely necessary and may result in an unhappy patient. Any gain in weight in a patient who has had congestive heart failure is almost certain to be caused by fluid, and it is never an encouraging sign.

The bedpan should not be used for cardiac patients, with the exception of those who are almost moribund, and then there is little need for it. The commode should be the receptacle of choice. The patient is moved so that his legs hang over the bed, he slides to his feet, pivots to the right or left as the case may be, and then sits down. This procedure can be carried out with a minimum of exertion.

Summary

The gastrointestinal symptoms associated with heart disease are important for two reasons: first, they cause the patient to seek advice regarding his stomach, and secondly, they mislead even the physician. As a result, unnecessary roentgenologic studies may be carried out on a patient who can ill afford to go through such procedures because of his cardiac or financial status. The recognition of the heart condition as the principal cause may save patients from unnecessary and dangerous surgical procedures. Perhaps even more important is the recognition of gastrointestinal lesions causing symptoms of heart disease; to label a patient as having cardiac disease is inexcusable without substantial evidence in both the history and the physical findings. Other causes should be sought, and in such cases a thorough survey is indicated.

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MEDICAL PROGRESS

ORTHOPEDIC SURGERY

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THE problems peculiar to the surgery of trauma have become the focal point of interest of a considerable proportion of medical men, regardless of prewar surgical training. It seems appropriate, therefore, to devote this report of progress in orthopedic surgery to injury and, because of their importance, to the infections with which injury is associated.

TRAUMA AND INFECTION

Infection frequently follows injury and may become of far greater importance than the injury itself. The many and diverse advances made in the field of surgical infection during recent years are often confusing to the clinician and difficult of evaluation. The mechanism by which traumatic infection is produced and develops — in other words, the pattern of infection — varies considerably with the predominant infecting micro-organism. The infection pattern of each of the pathogenic bacteria responsible for these infections is as a rule clearly defined. By it the course of an infection by a given bacterium may be anticipated and appropriate safeguards taken to arrest the local sepsis, prevent complications, and control toxemia. Accurate identification of the infecting bacteria, both aerobic and anaerobic, by culture of the tissue excised at débridement and of the wound exudate is essential to optimum surgical management. Sulfonamide therapy is now recognized as a deterrent to the spread of sepsis, effective through controlling bacterial invasiveness. Antitoxin therapy aids in the control of serious toxemias by neutralizing bacterial toxins absorbed from the septic focus. Increasing attention is being paid to nutritional factors involved in the resistance to infection. Aided by immunotherapy and the bacteriostatic action of the sulfonamides, surgical treatment of the local sepsis is taking on a broader scope. Earlier drainage of pus is now possible, as well as regional division of veins and excision of the septic focus. The immobilization of infected parts, long recognized clinically as of great importance, has been placed on a scientific basis. Débridement of open wounds, done at the earliest possible moment, stands as one of the best preventives of infection. Reports concerning the results of penicillin and gramicidin are promising, and development of the former in some of the hospitals of the armed forces is being followed with close interest.

Path of Entrance of Bacteria

In general, any injury such as contusion, laceration, compound fracture or an incised or penetrating wound may provide a suitable milieu for bacteria. The local conditions that favor infection are devitalization or necrosis of tissue, diminished blood supply, increased tissue tension resulting from tissue breakdown, hemorrhagic accumulations and foreign bodies — bits of clothing, dirt, shell fragments and so forth. Entrance of bacteria into these areas may be gained through air-borne contamination or direct inoculation, by lymphatic extension from a remote focus, such as a minute crack between the toes in epidermophytosis, or by the hematogenous route. The bacteria introduced by direct inoculation are usually those present on the clothing and skin at the site of injury, although they may be present on the inflicting instrument. The bacteria that are associated with wound infection may, almost without exception, be cultivated from the skin or clothing at various times, the number and type varying with the region injured, with environmental factors and with previous exposure to contamination. Wounds involving the inner recesses of the body, such as the respiratory and gastrointestinal tracts, may, in addition, be inoculated directly with the organisms that make the walls of these systems their natural habitat. All the available evidence points strongly to the conclusion that open wounds are infected by streptococci and staphylococci by subsequent contamination much oftener than by direct inoculation at the time of infliction. Of 355 wounds sustained under various circumstances — that is, industrial, street and miscellaneous accidents — Hare and Willits[1] found no Group A hemolytic streptococci within two hours of infliction, but grew members of this group from 4 of 108 wounds during convalescence under active treatment. The only exception to the above statement is in the Group A carrier class, because the skin and clothing of these patients may harbor such organisms in profusion. Of the 71 cases in

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which there were coagulase-positive staphylococci, 10 per cent showed a positive culture immediately following infliction, whereas 35 per cent did so during convalescence under treatment.

Contamination of wounds subsequent to infliction and during treatment is the most frequent source of wound infection. The practice of doing frequent dressings of open wounds on surgical wards is to be condemned. The air of these wards usually teems with pathogens introduced from wounds, dressings, bedclothing and the exhalations of other infected patients; from the dressing carriage and the breath and clothing of the attendants; and from the exhalations, skin and clothing of the patient. These organisms, often highly virulent, are blown about with the dust into every quarter of the ward by the constant coming and going of attendants, to settle in profusion upon the exposed wound surfaces. The results are often little short of disastrous. The clear recognition of this pernicious reinfection of wounds has led of late to the general adoption of closed-dressing methods, with incalculable saving in time, life, limb and function, and has eliminated a great deal of futile endeavor. It represents one of the major advances in the surgery of wounds. Open wound dressings should, therefore, be done not on the surgical ward but in a room set aside for the purpose. This room should be as free as possible of atmospheric commotion; adequate masks should be worn by everyone having anything to do with the wound, including the patient, and the dresser's hands should be prepared as for a clean surgical operation. Above these measures, the wound should be closed off from contamination at all other times. It should be exposed for dressing only when absolutely necessary, — for example, when there are signs of toxemia, increasing sepsis or complications, or saturation of the dressing, — and the dressing should then be done with the minimum of disturbance of the healing surfaces. The surgeon should plan at the time of operation to leave the wound in such a condition that only infrequent and nonmanipulative dressings will be necessary. Such a technic is now being stressed widely in civilian and military hospitals.

To the above there is a notable exception, namely, the Carrel-Dakin method of wound management. This superior technic is unfortunately fast becoming a lost art. Where complete facilities are available, and there is a surgical staff adequate in number and training, it undoubtedly has a definite place and will yield brilliant results.

Infecting Organisms

The bacteria commonly responsible for the infections that follow trauma are staphylococci (coagulase-positive), hemolytic streptococci, green-producing and nonhemolytic streptococci and clostridia. Anaerobic streptococci, enterococci and gram-negative bacilli (anaerobic) are less frequent wound pathogens. Statistical tables of wound contaminants naturally vary somewhat. In a bacterial analysis of a series of 200 fresh wounds that included lacerations, bullet wounds, dog and human bites, other puncture wounds, compound fractures, traumatic amputation and various automobile and other accidental wounds, Pulaski, Meleney and Spaeth[2] found the following distribution of organisms: *Staphylococcus aureus*, 52 per cent; hemolytic streptococci, 17 per cent; green-producing and nonhemolytic streptococci, 35 per cent; clostridia, 23 per cent; enterobacteria, 23 per cent; anaerobic nonhemolytic streptococci, 7 per cent; and anaerobic gram-negative bacilli, 2 per cent. The wounds contained on an average three different pathogenic species. This study also revealed that the older was the wound, the more anaerobes were present.

Bacteriology and Immunology

A brief résumé of the more recent contributions to the bacteriology and immunology involved in the common types of traumatic infection seems appropriate.

Staphylococci

Identification. Staphylococci are classified at present according to their virulence for man. Strains of *Staph. aureus* are almost without exception pathogenic; those of *Staph. albus* may or may not be. The most reliable test of pathogenicity is the capacity of culture filtrates to coagulate citrated, oxalated or heparinized plasma. Such coagulation is due to a soluble enzymal by-product, staphylocoagulase, common to pathogenic strains (Chapman et al.[3, 4] and Fairbrother[5]), and is now employed in Army, Navy and most civilian hospitals.

Toxic factors. Staphylococcal pathogenicity depends rather more on the intense local necrotizing action of the bacteria themselves than on their capacity to produce toxins. However, there are several known potent toxins.

Staphylococcal toxin (potent culture filtrate) produces the following clearly demonstrable effects: skin necrosis when injected intradermally, a rapid intravenous lethal action in animals when given in huge doses and hemolysis in animals (probably of minor importance in man). Leukocidin, long associated with filtrate toxin through its destructive or inhibitory action on leukocytes, is still difficult of technical evaluation, although it is thought to be of considerable importance (Blair,[6] Wright[7] and Lyons[8]). Hyaluronidase, an enzyme,

the "spreading factor" of Duran-Reynals, has been extracted from cultures of virulent strains. It markedly increases tissue permeability to bacteria (and dyes), intensifies bacterial invasiveness, and enhances infection. It is soluble, stable and non-specific (Duran-Reynals[9, 10] and McClean[11]). A separate and distinct toxin, enterotoxin, is associated with staphylococci responsible for outbreaks of food poisoning. In animals its potency when administered intravenously is much greater than that when given orally. In man the symptoms produced are vomiting and diarrhea, abdominal cramping pains and headache. These were symptomatic features in the fatal Bundaberg cases, and Wright[7] suggests this as the chief lethal factor in distinction to Blair's[6] idea of the filtrate toxin as the predominant cause of death in this disaster. Lastly, Stevens[12] has reported 3 cases of scarlatiniform rash occurring in *Staph. aureus* infection, implying an uncommon staphylococcal erythrogenic toxin.

Source. Coagulase-staphylococci are normal inhabitants of the human skin and nasal passages. There is evidence that their prevalence on the skin is not so great as is generally believed. Gillespie, Devenish and Cowan[13] found this group present on the skin of 19 per cent of 159 normal subjects, whereas Smith's[14] finding was 5 per cent of 100 persons. The same investigators found the organisms in the nasal passages of these people in 43 and 32 per cent respectively.

Pattern of infection. From the clinical standpoint, by far the most striking feature commonly seen in staphylococcal infection is the intense local necrotizing action of the organisms. It is as though the tissues in the immediate vicinity had been scorched. Inflammatory fixation is usually prompt, violent and complete, resulting in the formation of simple abscess (less frequently in carbuncle). The lymph channels are blocked with fibrin thrombi, whereby entrance into the lymphatic system is denied to staphylococci or their toxic products. The capillaries thrombose, and the infected area is walled off from the rest of the body (Menkin). All the evidence points to the assumption that staphylococci gain access to the thrombi formed in the vascular radicles, maintain themselves therein, and from these may enter the blood stream, in fragments detached from the infected thrombus. Expressed in clinical terms, microscopic septic thrombophlebitis may occur in the walls of the abscess. Lyons[8] has emphasized this fundamental step, as well as the frequency of staphylococci as the responsible organism in the bacteremia that results from it. Conveyed in the blood stream within septic emboli, staphylococci may gain access to various tissues according to a definite pattern that seems to be governed in part by infarction of the next capillary bed, in part by abscess of the infarcted area and in part by a thrombophlebitis that develops in the metastatic focus. Such metastatic lesions may in turn contribute to the blood stream further embolic contamination, whereby new foci develop. An intermittent bacteremia is thus set up. An abscess developing in an infarct infected with staphylococci may extend to the adjacent tissues or may rupture into a nearby serous cavity to produce a pleural or pericardial empyema.

The lesions that result from and contribute to staphylococcal bacteremia may include any of the following: pulmonary infarction, abscess, pneumonia, pleural empyema and pulmonary thrombophlebitis; visceral infarction and abscess; osteomyelitis; suppurative arthritis; thrombophlebitis of major veins; and brain abscess, thrombophlebitis and meningitis. To this list must be added staphylococcal endocarditis. The mechanism of valvular infection is as yet not defined. It is, however, well known that infected vegetations may become detached, contaminate the blood stream, and cause infarction and so forth. That these lesions comprise those found in metastatic staphylococcal infection is amply supported by autopsy findings. It is not the purpose of this review to discuss the clinical implication of each. The primary focus in these bacteremias may have healed and have been forgotten or may have escaped notice.

Such metastatic lesions vary in size from minute to massive, and may exhibit any part or all of the pattern. Thus, a pulmonary infarction may escape detection or may not occur, yet a positive blood culture attests to the passage of the bacteria through the lungs. On the other hand, infarction may be massive and promptly fatal, or the lesion may suppurate and become fatal later. Variations in local tissue resistance may explain many of these modifications in pattern. However, a suppurative focus in staphylococcal bacteremia is constant. When general infection becomes established, with numerous and extensive metastases and a constant embolic contamination of the blood stream, there is usually a fatal termination.

Exceptions to the above pattern must occur in the nature of things. These are noteworthy but do not alter the validity of the conception. Staphylococci may reverse the usual order and take on some of the attributes of streptococci to produce cellulitis, lymphangitis and a scarlatiniform rash.

Management. The advent of the sulfonamides has not lessened the importance of drainage of staphylococcal pus. The infected area should be exteriorized by adequate drainage, once the inflammation has become fixed locally. In severe infections every effort must be made to improve

the general condition of the patient by sedation and intravenous fluids, and by transfusion, if indicated, while the involved tissues are localizing the infection. Chemotherapy is begun at once. By this means bacterial invasiveness is held in check until natural resistance, aided by appropriate surgical measures, is adequate. Often valuable time may be gained by administering the sulfonamide (except sulfanilamide) intravenously in the form of the sodium salt (prepared capsules). It is essential that the infected part be immobilized by adequate splinting, as discussed later.

In spite of proper and adequate drainage, chemotherapy, splinting and ideal general care, metastatic infection through septic embolism may occur, and vigilance is required to detect intravascular infection at its inception. Such infection may be heralded by a chill, a temperature spike of 103°F. or over and a leukocytosis in excess of 15,000. A sustained high white-cell count, a continued fever, repeatedly positive blood cultures, a palpable thrombosed vein and a pulmonary infarct offer positive evidence of its persistence. Prompt measures directed to control this factor are indicated. Excision of the primary focus when feasible may be successful, or the implicated vein, if accessible, may be excised or ligated.

Once established, metastatic infection must be dealt with according to the same principles employed in the management of the primary focus. Evidence is beginning to appear that penicillin is a highly effective bacteriostatic and bactericidal agent in staphylococcal as well as many other infections. It is not yet available for civilian use.

Efforts to induce staphylococcal immunity in man by antitoxin, toxoid, antibacterial serum, bacteriophage or transfusion have not to date been successful in notably altering the course of infection. Lyons[8] attributes the improvement claimed for these in reducing the mortality rate in staphylococcal bacteremias to an uncritical selection of cases or to a nonspecific shock reaction enhancing resistance. A true staphylococcal bacteriolysin — excepting possibly penicillin — has not yet been demonstrated.

Aerobic streptococci. The classification of streptococci by serologic methods has relieved much of the confusion that attended the general problem of streptococcal infection and complicated the many questions it raised. Thus the limitations of antibacterial serum therapy can be pointed out, sources of infection can be accurately traced, and the demonstration can be made that any of the various types of streptococci virulent for man may produce a given specific streptococcal clinical picture, such as scarlatina, erysipelas, septic sore throat and so forth. Lancefield, using extracts of the coccal bodies, has divided the streptococci on the basis of precipitin reactions into thirteen groups — A through M. Each group differs serologically from the others on the basis of a group antigen, C substance, a carbohydrate. Each group is divisible into a number of types on the basis of its capsular material — M substance, a protein — and each includes hemolytic (β), green (α) and nonhemolytic (γ) dissociants. Griffith has derived a similar type classification by using a slide-agglutination technic. Group A (Lancefield) or *Streptococcus pyogenes* (Griffith) is responsible for 95 per cent of serious streptococcal human infections. Group A has been shown to contain at least twenty-nine types. Groups B, C, D, F, G and H may also cause human infection.

Toxic factors (Zinsser,[15] Keefer[16] and Wright[7]). Group A hemolytic streptococci may produce a number of toxic substances. Erythrogenic toxin (A and B), when occurring in large amounts in sensitive persons, gives rise to a scarlatinal rash, fever, vomiting and a rapid pulse. A hemolytic toxin, hemolysin (streptolysin), can be demonstrated, but the part played by this unstable toxin in streptococcal infection is not clear. A fibrinolysin appears to dissolve human fibrin and inhibits its formation from fibrinogen. Both the hemolysin and fibrinolysin are neutralized by streptococcal antitoxin. A leukocidin is also found in streptococcal filtrates that destroy leukocytes in vitro. It is antigenic and type specific. Hyaluronidase (Duran-Reynals's "spreading factor") is present in toxic filtrates.[11] It greatly increases tissue permeability and enhances the invasiveness and virulence of the organisms.

Source. Human, Group A, hemolytic streptococci are normal inhabitants of the lymphadenoid tissue of the throat. A considerable proportion of people are carriers of this group. The organism is found in the throat most commonly in winter and the early spring months, particularly in those who have been in contact with patients with acute streptococcal throat infection, especially if they have "colds." Such persons should scrupulously avoid operating rooms and attendance on wounds. It is generally known that Group A streptococci are rarely found on the skin.

Pattern of infection. Infection with hemolytic streptococci (Group A) manifests itself in a variety of ways. Two factors come into play in all these: toxic and septic.

The toxic manifestations are fever, rapid pulse, headache, malaise, prostration or shock, nausea or vomiting and often erythema.

The many clinical septic reactions reveal a considerable similarity. A rapidly spreading cellulitis

(phlegmon) is one of the outstanding characteristics, in sharp contrast with the rapid local fixation (simple abscess) accorded staphylococcal infection. This occurs as a widening area of induration, often erythematous. It marks the progress of the local infection, and is produced by edema, hyperemia, phagocytic cellular infiltration and vascular thrombi. Bacteria may be found throughout. The margins merge abruptly into the normal tissue without early barrier formation. Suppuration may occur and is found in a diffuse thin layer, or it may be more or less localized, as seen in staphylococcal infection. Fixation may not occur for forty-eight hours, during which time the infection may have spread, for example to involve almost the entire thigh or the full length of a limb.

An equally conspicuous characteristic of the streptococcal pattern is the selection by these organisms of certain tissues for their depredations. Such tissues include the following: the lymphatics (and vascular endothelium in general); the fasciae; the cavities with mesodermal epithelial lining, that is, tendon sheaths, joints, bursas and the peritoneum, pleura and pericardium; and the lungs.

The infection of lymphatics may be limited to the local reticular (capillary) portion of the lymphatic system around the wound, producing induration and, if the skin is involved, blushing or erythema. Extension from the lymphatic reticulum may occur into the adjacent tissues, producing cellulitis, with or without suppuration; infection of the reticular lymphatics of the skin in a highly virulent form constitutes erysipelas. Or the main lymph channels may become involved, either directly from a wound more or less appreciably infected or by extension from reticular infection. In the superficial pathways this involvement is revealed by the typical red streaking of lymphangitis; in the deep pathways, the indurated channel may be detected by palpation as a tender cord, especially if grease is applied to the skin to reduce friction (Lyons[17]). Extension into adjacent tissues may occur, producing cellulitis and suppuration, and if the intimate anatomic relation of veins and lymphatics is recalled, adjacent phlebitis and thrombophlebitis (Homans[18]) is not surprising. Lymph-node infection occurs, with or without appreciable lymph-channel involvement, but the portal of entry is usually readily demonstrable. Suppuration of the nodes may ensue; again, there may be extension into adjacent tissues in the form of cellulitis, with or without suppuration (Frank[19]).

Streptococcal infection in a fascial plane may sweep quickly along the deep fascia, usually parallel with the regional body surface, producing a typical cellulitis. Edema may be striking, and may clearly demarcate the limits of the infection. As the sepsis progresses, erythematous areas may appear and suppuration may develop. The process may be somewhat limited by the fascial compartments. As in most streptococcal invasions, the condition is a grave one, and lymphatic, thrombophlebitic and metastatic complications may arise. The condition may be blood borne following contusion. Hemolytic streptococcal gangrene (Meleney[20]), termed "gangrenous erysipelas" by the older writers, is essentially a virulent streptococcal cellulitis of the subcutaneous tissues with secondary skin involvement, and leads to massive gangrene. Toxic and septic complications add to the seriousness of the picture.

Infection of tendon sheaths, joints, bursas, pleura and so forth by hemolytic streptococci is frequent and may occur by direct inoculation, by extension from a neighboring focus or via the blood stream. Metastatic joint infection is not uncommon.

Bacteremia occurs when hemolytic streptococci reach the blood stream via the lymphatics, either directly or by extension through thrombophlebitic foci. Clearance is usually prompt unless bacteria continue to pour in. Cardiac valvular lesions may result and in turn provide further contamination from detached vegetations, as in staphylococcal bacteremia. Blood-borne streptococci are prone to metastasize to the lungs, — infarction and bronchopneumonia, — to the joints, — especially in children, — to bone in infants and to the subcutaneous tissues. Metastatic lesions follow the same general pattern of infection as do the primary lesions. They may in turn give rise to further metastases, and if these repeat the process, and infection becomes overwhelming, death finally ensues. A fatal termination may, however, occur with great rapidity in people of subnormal resistance attacked by great numbers of organisms of extremely high virulence.

Streptococci are destroyed in the body by phagocytosis and intracellular digestion, and in this, the macrophage and histiocyte are more effective than the polymorphonuclear leukocyte.

Management. The bacteriostatic action of the sulfonamides has proved to be of enormous value in these infections. By their use time is gained for the patient to marshal the natural forces of resistance and to fix the infection, and for the surgeon to institute most effectively the appropriate measures to improve the general condition and to combat the local sepsis. Complete bed rest in a hospital, sedation, maintenance of body fluids by intravenous saline solution and transfusion, if indicated, are standard general measures. For the local condition, rest by immobilization through appropriate splinting is of the first importance, as discussed below. Hot, moist dressings maintained

over the affected areas are of generally accepted value. Vigilant attention to the local sepsis and alertness to detect complications are essential. In the ordinary form of streptococcal cellulitis — and in erysipelas — surgical drainage should be deferred until suppuration occurs, and should then be restricted to the suppurative zone. In hemolytic streptococcus gangrene, on the contrary, longitudinal incisions are made through the involved tissue and are extended just beyond the margins into healthy tissue as soon as the diagnosis is established. Dakinization of the incisional wounds may then be employed to advantage.

The complications of hemolytic streptococcus sepsis are variously dealt with. Thrombophlebitic foci are blocked off by ligation or excision of the appropriate proximal vein, if it is accessible, and areas of suppuration are drained by incision confined within the suppurative zone. Joint infection is widely treated by repeated aspiration and immobilization; pulmonary, pleural and peritoneal infections are treated by appropriate special local measures.

Streptococcal antitoxin, although it neutralizes the erythrogenic toxin, has a limited therapeutic sphere in surgical infections. It may be helpful in cases in which the signs of intoxication are severe, but it does not alter the septic process. Scarlet-fever convalescent serum gives fewer reactions of serum sickness, but it is less potent than the antitoxin. Recently, the purification of antitoxin has considerably lowered the incidence of serum reactions. At present the only method available for supplying specific antibacterial substances in hemolytic streptococcus infections is Lyons's[17] immune-blood transfusion. It aids in clearing the blood stream of organisms and in localizing the infection, and is applicable to patients with bacteremia. A trained person is required to select the proper donor, that is, one compatible and possessed of sufficient type-specific opsonizing antibody. It has been demonstrated that, at the present time, all commercial *antibacterial* streptococcal serums are valueless, if not actually harmful.

(To be concluded)

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MASSACHUSETTS MEDICAL SOCIETY

PROCEEDINGS OF THE ONE HUNDRED AND SIXTY-SECOND ANNIVERSARY

May 24, 25 and 26, 1943

THE one hundred and sixty-second anniversary of the Massachusetts Medical Society was observed in Boston, on Monday, Tuesday and Wednesday, May 24, 25 and 26, 1943, at the Hotel Statler.

Twelve hundred and seventy-five physicians were registered.

Monday, May 24

The supervising censors met at 5:00 p.m. in the Hancock Room.

The Cotting Supper was served to 179 councilors in the Salle Moderne. The annual meeting of the Council was held in the Georgian Room at 7:00 p.m. There were 204 councilors in attendance.

Tuesday, May 25

The first general session was opened by the president, Dr. George Leonard Schadt.

Dean Edwin J. Van Etten offered prayer.

Dr. Peirce H. Leavitt and Dr. Leroy E. Parkins acted as chairman and co-chairman respectively. The attendance was 500.

This session took the form of a symposium on burns with special reference to the Cocoanut Grove disaster.

The one hundred and sixty-second annual meeting of the Society was held at 11:00 a.m. in the Georgian Room. Dr. George Leonard Schadt presided. The attendance was 350.

Dr. Schadt spoke at length on the state of the Society. The Annual Oration, "Small Puddles," was delivered by Dr. Edward P. Bagg at the close of the meeting. During the course of the meeting the President introduced Dr. John F. Holmes, of Manchester, delegate from the New Hampshire Medical Society.

The annual luncheon was served in Parlors A, B and C to 151 fellows. Dr. Schadt presided, and Reverend Leo G. Burke, O.M.I., offered grace and benediction.

A clinical meeting was held in the Georgian Room from 2:00 to 5:00 p.m. under the chairmanship and co-chairmanship of Dr. Roger I. Lee and Dr. Dwight O'Hara respectively. The attendance was 550.

In the late afternoon, Mrs. George Leonard Schadt held a tea at the Harvard Club in honor of Mrs. Roger I. Lee and Mrs. Elmer S. Bagnall. Forty ladies were present.

The annual dinner was held in the Georgian Room at 7:00 p.m. with 325 in attendance. Rabbi Samuel J. Abrams said grace.

The speaker at the dinner was Mr. James A. Hamilton, of New Haven, president of the American Hospital Association, professor of hospital administration at Yale University and director of the New Haven Hospital.

Dr. George W. Thorn, Hersey Professor of the Theory and Practice of Physic, Harvard Medical School, and physician-in-chief, Peter Bent Brigham Hospital, delivered the Shattuck Lecture. His subject was "Physiologic Considerations in the Treatment of Nephritis."

Wednesday, May 26

There was a general clinical meeting in the Georgian Room from 9:00 a.m. to 12:00 noon under the chairmanship and co-chairmanship of Dr. James W. Manary and Dr. Edward J. O'Brien, Jr., respectively. The attendance was 550.

From 12:00 noon until 2:00 p.m. there was a series of section meetings and luncheons.

The Section of Medicine held forth in Parlors B and C under the chairmanship of Dr. Laurence D. Chapin. The attendance was 44.

The Section of Surgery met in Parlor A, under the chairmanship of Dr. Oliver Cope. The attendance was 40.

The Section of Pediatrics met at the Junior League, Zero Marlboro Street, under the chairmanship of Dr. James M. Baty. The attendance was 33.

The Section of Obstetrics and Gynecology met at the Women's Educational and Industrial Union, 264 Boylston Street, under the chairmanship of Dr. Christopher J. Duncan. The attendance was 51.

The Section of Radiology met in Room 404, Hotel Statler, under the chairmanship of Dr. Joseph H. Marks. The attendance was 22.

The Section of Physiotherapy met in Parlors D and E, Hotel Statler, under the chairmanship of Dr. Henry A. Tadgell. The attendance was 20.

The Section of Dermatology and Syphilology met in the Hancock Room, Hotel Statler, under the chairmanship of Dr. J. Harper Blaisdell. The attendance was 36.

From 2:00 to 5:00 p.m. a general clinical meeting was held in the Georgian Room under the chairmanship and co-chairmanship of Dr. Charles E. Mongan and Dr. Frank R. Ober, respectively. The attendance was 450.

There were 63 technical and 16 scientific exhibits.

The motion pictures attracted the attention of a large number of fellows.

The lists of admissions and deaths are appended, together with the official lists of officers, standing and special committees, councilors, censors and so forth.

MICHAEL A. TIGHE, *Secretary*

ANNUAL MEETING OF THE SOCIETY

The one hundred and sixty-second annual meeting of the Massachusetts Medical Society was called to order by the president, Dr. George Leonard Schadt, in the Georgian Room of the Hotel Statler in Boston at 11:00 a.m., May 25, 1943. There were approximately 350 fellows present.

The Secretary offered the record of the last annual meeting as published in the *New England Journal of Medicine*, issue of July 9, 1942, and moved its adoption. This motion was seconded by a fellow, and it was so ordered by vote of the Society.

Dr. Schadt presented Dr. John F. Holmes representative of the New Hampshire Medical Society. Dr. Holmes responded as follows:

> I am very pleased to be here with you today, and I congratulate the Society on the wonderful program that has been presented this morning and the program that has been arranged for the rest of the meeting. It is a pleasure to be here, and I feel highly honored.

No delegates were listed from Maine, Vermont, Rhode Island or Connecticut. It was explained by the Secretary that these states were not represented owing to the exigencies of the War.

The Secretary announced that the membership of the Massachusetts Medical Society, as of May 26, 1942, was 5708, that the Society has gained during the current year 136 new members and 17 restorations, that it has lost 76 members by death, 16 by resignation and 16 by deprivation of their membership. He added that the net gain has been 45 and that the membership as of May 25, 1943, was 5753.

At the request of the President, the members stood for one minute in silent tribute to the memory of those fellows who had gone beyond.

At that point Dr. Peirce H. Leavitt, vice-president, assumed the chair. Dr. Schadt addressed the Society as follows:

> Since our last meeting we have lost a valuable and honored servant of the Massachusetts Medical Society in the person of Dr. Charles Shorey Butler. Dr. Butler had been treasurer of the Massachusetts Medical Society, I believe, since 1931, approximately twelve years. He gave a great deal of himself to the Society, which he loved dearly. After many years of faithful service, he continued in office when the requirements of his health should have demanded a rest. He was finally prevailed on to ask for an indefinite leave of absence, which was granted. Dr. Butler died February 23, after an illness of several months. He had a peaceful death, and one that ended much suffering.
>
> It was my privilege to know Dr. Butler intimately and to love him. He was a most kind and able man, one of the gentlemen of the old school, who, unfortunately, are passing from this scene.

At the request of Dr. Schadt the members stood for one minute in silent tribute to the memory of Dr. Butler.

It was moved and seconded that the President appoint a committee to bring in suitable resolutions on the death of Dr. Butler. It was so ordered by vote of the members.

The Secretary asked the district secretaries to aid his office in gathering the list of fellows who have entered and who will enter the service of our Country. Dr. Schadt seconded this request and called on the district presidents to help in this effort.

Dr. George L. Schadt delivered the presidential address on the state of the Society. He spoke as follows:

> It is a pleasant duty for each president retiring from office to submit at the annual meeting a report on the state of the Society. This I shall do now.
>
> I am very much pleased to be able to tell you that the state of the Massachusetts Medical Society at the present moment is excellent. This I hasten to assure you is not due to anything I have done, but due entirely to the unselfish work of the other officers, particularly Dr. Peirce H. Leavitt, vice-president, Dr. Michael A. Tighe, secretary, and Dr. Eliot Hubbard, Jr., acting treasurer, and of course to the chairmen and the members of the various standing committees and the appointed committees during the year. I have learned anew — and it has been a very refreshing thought, too, — that when one wishes anything done one should ask a busy man. He will always have time to do it. I have asked many busy doctors during the year to accept appointments on committees as chairmen and as members, to carry out difficult and tedious assignments, and I am happy to say I did not have one refusal.
>
> As has already been stated, the Massachusetts Medical Society is in excellent condition, both physically and

financially. Starting my year as your president, it was with the determination that we should have a peaceful, constructive year — a year free from petty and unprofitable squabbles. I think I am correct in saying that in the past we have had some petty squabbles and have always found them unprofitable. Your president has enjoyed the duties of his office very much. He has had the very happy privilege of making fifteen official visits to fourteen of the eighteen district medical societies. During these visits he has had the privilege and pleasure of meeting many of the members, and he hopes that he has made friends among them. It is a delightful experience to visit the different district societies, to talk with the officers and the members, and to learn the problems they have, — and it may be said these problems vary in the different sections of the State. Many of these problems, too, are local ones, and should be handled locally, and your president wishes to say that he has discovered that these problems are being handled in an excellent manner.

In going about among the various district societies, I have endeavored to tell the members what the Massachusetts Medical Society is doing, what some of the problems are that have arisen, to answer any questions the members may have, and also to gain the good will and support of the membership as a whole.

The late Dr. Burrage, our very efficient secretary for many years, wrote a most interesting and instructive history of the Massachusetts Medical Society, bringing it up, I believe, to 1926. Dr. Burrage left a number of volumes to the Society, requesting the secretary to present to each president during his term of office a copy of the book. Dr. Tighe presented me with my copy, and may I say I have read it from cover to cover and enjoyed it tremendously. I urge the members of the Society to read this history of the oldest medical society in the United States in point of uninterrupted meetings from the time of its beginning, in 1781, to the one hundred and sixty-second anniversary, in 1943. I cannot refrain in leaving this subject from expressing the hope that some historically minded member of the Society will carry on this history from the point where Dr. Burrage left off, — the year 1926, — bringing the history up to date.

May I now extend my heartfelt thanks and appreciation to all the officers with whom I have worked during the year and to all committee chairmen and committee members. I shall always have the deepest appreciation for what they accomplished for me during the year. It is not my purpose to mention many names in this report, but I do not believe the report would be complete without speaking of several.

The interest and the work of Dr. Michael A. Tighe, our secretary, is deserving of the gratitude of every member of the Society — a grand gentleman and a born secretary. Frankly, I find it difficult to understand how it would have been possible to have carried on without him. He knows every detail of what is going on in the Society, and his good advice and his assistance are always readily available. I wish at this time to extend to him my affectionate regard and my thanks for his help during this year.

May I also express to Dr. Eliot Hubbard, Jr., our acting treasurer, my deep appreciation for his help during this rather trying year. Dr. Hubbard stepped into the breach when our late treasurer, Dr. Charles S. Butler, found it impossible to continue with his duties, because of his health. Dr. Hubbard rather reluctantly assumed the office of acting treasurer. He gave unselfishly of his time to the work, and on Dr. Butler's death took over all the duties of the office. Dr. Hubbard has steered the financial ship of state most capably, and though we shall all miss Dr. Butler, for whom I am sure we all had a great affection, we may feel that in Dr. Hubbard we have a worthy successor.

Dr. Charles Shorey Butler, the treasurer of the Massachusetts Medical Society since June 9, 1931, passed away on February 23, 1943. Dr. Butler served the Society as treasurer for approximately twelve years. His devotion to the Massachusetts Medical Society was complete. He gave unselfishly of his time and of himself in guarding its financial resources. He spent much time investigating investments that would make secure the principal. He was a lovable man — a man of whom it may well be said, — paraphrasing a remark by Oscar Wilde, — ". . . a charming gentleman, he had no enemies; he had many friends, and everyone loved him." It was my privilege to know Dr. Butler intimately, and knowing him was to love him. He most reluctantly and only after much urging relinquished his duties, and then only when the state of his health made any other step impossible. In the death of Dr. Butler the Massachusetts Medical Society has lost a rare personality, an ideal treasurer and a friend.

The Society, as already stated, is in sound condition. It is strong, vigorous, virile. During the year it has conducted much constructive work, of which more will be said later. The Society has grown in membership, and as will be noted from the figures given herewith — we have increased in membership steadily in the last five years:

June 8, 1939	5438
May 22, 1940	5542
May 22, 1941	5639
May 26, 1942	5708
May 18, 1943	5752

These figures, of course, include members now in the armed services, but members nevertheless of the Massachusetts Medical Society. It seems to me the outstanding feature of these figures is the fact that during this tremendous cataclysmic war the Society has grown.

You will be interested, I am sure, to know — and I received these figures from Dr. Reginald Fitz, of Suffolk, at a recent meeting of the State Committee of the Procurement and Assignment Service — that according to the records of the *American Medical Directory* there were 8085 physicians in Massachusetts in 1942. Of this number 2734 were not members of the Society, and of this group 1269 are in the armed services. On the other hand, 5351 were members of the Massachusetts Medical Society. You will note an apparent discrepancy between these figures and the figures I have given you above, but since the directory is published only every other year there is, naturally, a lag in the figures. With reference to the number of members of the Society in the armed forces, I am informed that to date there is a total of 1256. Thus, a total of 2525 doctors from Massachusetts are members of the armed services — the Army, the Navy, the Marine Corps and the Public Health Service.

I am glad to say that the Council meetings held during the year have been well attended. There is one fly in the ointment, however: although many councilors attend the morning session, far too many leave at the intermission or directly after the Cotting Luncheon. As a matter of fact, there have been a number of occasions in past years and during this year that at the afternoon meeting

there has barely been a quorum, so that business might be transacted. As you and I know, the Council is the deliberative body of the Massachusetts Medical Society; its membership is based on the premise that every councilor represents twenty members of the district from which he comes. Therefore, when a councilor does not attend a Council meeting or attends only part of the meeting, twenty members have no representation whatever or have only partial representation. In my opinion, the districts should be most careful in the selection of their councilors, electing those gentlemen who realize the importance of the position and will give up the three days necessary to come to Boston and to attend the Council meetings from beginning to end. It might just as well be said here that some of the councilors living in Boston and towns adjacent not only do not attend the Council meetings, but when they do attend leave at noon or shortly thereafter. This problem has been discussed at the Executive Committee meetings, and at the annual meeting of the Council it was moved and voted that the meetings in the future begin at 10 in the morning and continue without interruption to 2 in the afternoon, or until all business is transacted. I am sure that following this rule we shall have a quorum of the Council at every session.

The Committee on Membership has been most active and has done a splendid job. During 1942 and 1943 it received a total of 95 membership applications, of which 65 were approved, 11 were disapproved, 9 were disapproved by the district committees, and 16 were held for further information. It is most interesting to note that there was close agreement between the central committee and the district committees, in that there was only a difference of two in the number disapproved by these committees.

The Committee on Membership has recommended to the Executive Committee that the question of establishing a junior membership in the Massachusetts Medical Society be postponed for the time being. The question whether the Society should establish a junior membership was brought to the attention of Dr. Tighe, our secretary, during the year. We discussed the question and then brought it to the attention of the Executive Committee, which recommended that it be passed on to the Committee on Membership. It may be that at some time in the future junior memberships will develop into a worthwhile project in the Society.

The State Committee of the Procurement and Assignment Service has done an excellent piece of work under difficult and trying conditions. It was my privilege to sit in at most of the meetings, and I was also asked to go to Washington on a number of occasions to attend meetings presided over by Dr. Frank Lahey, chairman of the Directing Board of the Procurement and Assignment Service. These committee meetings were very much worth while. I am informed by Dr. Lahey, in a letter dated April 24, 1943, that approximately 2300 doctors from Massachusetts are in the armed forces, of whom 1200 are members of the Massachusetts Medical Society and 1100 are not. Again it should be mentioned that there is an apparent discrepancy between these figures and some figures given earlier in the report. The discrepancy is apparent only because the figures were arrived at on different dates and under different circumstances. The quota for Massachusetts in 1943—and I have seen these figures recently—is 33 per cent complete up to April 27. Five hundred and ninety more physicians will be needed from Massachusetts before the end of the year, and the majority of these physicians will have to be taken from the large centers, that is, from the large districts—Suffolk, Middlesex South, Norfolk, and other districts in and about Boston. Massachusetts, together with New York and Connecticut, has the unenviable reputation of being one of the three states that have not filled their quotas for 1942, the percentage being 90 per cent. It is not necessary, I am sure, for me to tell you of the urgent need for physicians in the armed services. In going about the State and in visiting Washington, Chicago and other cities, I have heard all the arguments pro and con. I have heard arguments about essentiality, until frankly I am fed up with the word. In my opinion, the word "essential" is the cause of more trouble in procuring the adequate number of physicians than any other one word or one thing, and I for one should like to see the whole question of essentiality ruled out. In my opinion, it would be a very constructive thing and would help immeasurably in getting many physicians to enter the services who now are hesitating for one reason or another, if every doctor of service age, that is, under forty-five or fifty, could be put in uniform on active service.

The Committee on Ethics and Discipline has had a busy year, and under its very able chairman has done a fine piece of work. Your president has had the privilege of attending all its meetings. It is somewhat discouraging to listen to some of the problems that are brought up to this committee and to realize that there are men practicing our profession who apparently are not guided by the principle annunciated many years ago: "Do unto others as you would have them do unto you." Some of the cases presented have demonstrated a sordid viewpoint and greed, and others lack of appreciation of the ethics of medicine under which we practice in this enlightened age in the State of Massachusetts. May we hope that as the years go on there will be less and less of this in our profession, and less and less need for problems of this kind being brought before this committee.

In speaking of the Treasurer's report, may I again express to Dr. Hubbard, our newly elected treasurer, my deep appreciation for his help during this trying year. Dr. Hubbard informs me that our finances are in excellent condition. We have, I believe, approximately $220,000 in bonds. Why the Society has this much money I do not know, and have never been able to find out to my satisfaction. It is my distinct impression that this money should be at work doing good deeds in the way of scholarships or in some other manner that the proper committee might well deem desirable. Dr. Hubbard further informs me that to date he has collected $41,493 in dues, but that there are still 706 members whose dues remain unpaid, totaling $7060. When this amount is received our treasurer will have collected $48,553. I am quite sure that when the second bill goes out the members who have not paid will promptly do so; they will then receive the *New England Journal of Medicine* and will know that they are pulling their weight in the boat.

The *Journal* has continued its successful career under the able and brilliant editorship of a gentleman whose name you well know, and under the direction of a very able committee. You will, I am sure, be interested in the figures given herewith, showing the increase from year to year of subscriptions to the *Journal*, in 1943 totaling 10,347:

Subscribers	1943	1942	1941	1940	1939
M. M. S.	4202	4740	4873	4844	
Regular	4494	3281	3168	2698	6305
Student	1651	696			
Totals	10347	8717	8041	7542	6305

This includes an order on May 1 for 541 subscriptions from the Surgeon General's Office, for distribution to all Army hospital installations of 25 beds or more.

During 1942, the *Journal* received 1984 new "outside" subscriptions, a total that is over 50 per cent greater than the total received in 1941. Of these subscriptions, 1085 were from regular subscribers and 899 from medical students. As would be expected there were many cancellations, undoubtedly owing to enlistment in the armed forces; the net increases were 216 regular subscribers and 711 medical students, which total 927. (As of December 31, 1942, journals were being sent to 5083 members of the Society (a decrease of 74 from the 1941 figure), 3462 regular subscribers and 1193 medical students, a total of 9738, which is greater by 853 than the total for 1941. This does not include 348 members of the New Hampshire Medical Society, who receive the *Journal* once a month.

The editorial board took action on 238 manuscripts during 1942, a decrease of 90 compared with the figure for 1941. Of these papers, 169, or 71 per cent, were accepted.

Operations for the year resulted in a net loss of $17,550, which is approximately $2450 less than the amount appropriated by the Society. The net amount received from the Society was $19,100 the balance, approximately $1550, being covered by an increase in current assets. The net cost per member of the Society was $3.43, as compared with $3.20 in 1941.

There was an increase in operating expenses due to the larger circulation, the increased cost of binding and mailing and the fact that in 1942 fifty-three issues of the *Journal* were distributed, which happens every seven years when January 1st falls on a Thursday.

It is difficult to predict what will happen in 1943. Undoubtedly the number of member subscribers will be decreased owing to their enlistment in the armed forces; on the other hand, it seems likely that the number of "outside" subscribers will hold its own or possibly show a slight increase in spite of cancellations. Expenses should decrease as a result of lower publication costs (provided the total circulation is smaller) and an increase in advertising revenue, owing to higher rates, which became effective on January 1. The decrease in the number of manuscripts submitted for publication may become a serious problem. In any event, the pagination of the *Journal* will be cut down since the members of the editorial board believe that the high standards for acceptance should not be lowered.

I am sure you will all agree with me that we may take deep satisfaction in having an exceptionally excellent journal, one that ranks very high throughout the Country.

As most of you already know, the Society has within recent years been carrying on under a budget system. The Committee on Finance, the chairman of which has given unselfishly of his time on this work, has prepared within recent years a very complete budget, and the Society has been living within this budget. Anyone who has had any experience at all in preparing budgets and living within them knows the difficulties that develop. However, I may say that we are living within our budget, and that notwithstanding a decrease in revenue due to members leaving for the armed services.

Within recent years there have developed within the Society several district-society journals. At present there are three: one published by the Worcester District Medical Society; one published by the Norfolk District Medical Society; and one published by the Hampden District Medical Society. It is my studied opinion that the development of district medical society journals is a good thing for the Society, provided their activities are restricted to the district society that they represent. It is well, certainly, for the larger district societies that there be some medium through which the news of the district may be transmitted to its members from time to time: what the members are doing, what the hospitals in the district are doing or may have to offer in the way of clinics to the membership, and any other items that may be of local interest. I do not believe, however, that these journals should have a circulation larger than its membership. I am of the opinion that if and when the circulation of a district journal goes beyond that of its membership, then we may well look for and expect difficulties of one kind or another. Certainly these journals should not expect to compete with the *New England Journal of Medicine*, our own journal. Whereas the latter covers a much larger and a more comprehensive field, the former should, as already noted, devote their efforts to the county societies. I am rather hopeful that some of the larger district societies will see fit to publish journals, and it seems to me, too, that two, three or four adjacent smaller districts might well co-operate in publishing a journal that would cover their respective districts.

As I have gone about the State visiting the various district medical societies, I have taken the opportunity of pointing out the fact that the Massachusetts Medical Society as constituted today is practically completely decentralized, in that the Society is in the hands of its members, and not in the hands of any group in or about any of the districts or in or about Boston. Today we have three large state-wide committees. These committees have a duly elected member from each of the eighteen district societies. Besides this, the officers are all members *ex-officiis*. These committees, as you know, are the Executive Committee of the Council, the Committee on Public Relations and the newly organized Committee on Legislation. The work of each is important, and having attended practically all the meetings of these three committees this year, I can assure you that much constructive work was done in behalf of the Society.

I should like to emphasize at this time a point I have made in talks to the various districts. It is this: any member of the Massachusetts Medical Society may at any time gain a hearing in the Council by bringing to the attention of one of the councilors from his district the subject he wishes discussed. His subject will be received and considered. If found worth while it will be accepted. In other words, I wish to re-emphasize the fact that the Massachusetts Medical Society today is a society of approximately 6000 doctors, and is, as Abraham Lincoln so well said: "A government of the people, for the people and by the people."

With reference to the Executive Committee of the Council, this committee has worked hard all year, and since the amount of work crossing Dr. Tighe's desk is increasing each year, the subjects and problems to be considered are many and diversified. This committee as originally planned and set up was organized to lessen the detail of work brought before the Council and to shorten the Council meetings. Unfortunately this desired re-

sult has not been achieved as yet, but we are quite sure that it will be in the years to come.

The Committee on Public Relations has held regular meetings and has discussed many very important problems — problems with which you are all familiar, I am sure. One of the most important of these, of course, was the work of a subcommittee meeting with Mrs. Emma Tousant, chairman of the Industrial Accident Board. As the result of these co-operative meetings, considerable constructive work has been achieved in bringing about a better approach to the care of the injured workman, and a more equitable treatment of the doctor on the individual case.

The Committee on Legislation is the last of these large committees organized — as a matter of fact, it was only organized this year, but has already done a splendid bit of work. I am happy to be able to report that without lessening the scope of its work, this committee was able to return to the treasurer $1450 of its budget.

We are ending this year under a new set of by-laws. The committee delegated to revise the by-laws did an excellent piece of constructive work, and although we all did not see eye to eye with them, I am happy to say that the by-laws have worked out well, and with practically no confusion. One of the outstanding changes was that the collection of dues was placed in the office of the treasurer of the Society, instead of through the treasurers of the district societies. This collection of dues has worked out quite satisfactorily, and I am certain that in the years to come all the wrinkles will be smoothed out. It has, of course, meant a lot of work for the treasurer and his office, but he has done a splendid job and deserves much credit. As stated elsewhere in this report, there are still 706 members who as yet have not paid their dues. I am quite sure, however, this has merely been an oversight and that soon all their dues will have been received.

We have had several new committees that have done excellent work. One of these is the Committee on Industrial Health. This committee, under the able chairmanship of one of our most distinguished members, has carried out an extremely constructive program. The committee has held two large meetings, both at the Harvard Club, one in November and the other the latter part of April. Both these meetings were well attended — the first having an attendance of around 300, and the latter 160. Both programs were excellent. One of the most gratifying features of these meetings was the caliber of the speakers and how very ready each of these distinguished gentlemen was to accept the committee's invitation to appear on the program and to bring to the members of the Society up-to-date viewpoints on the various problems of industrial health. This committee will, of course, continue its work, and I believe plans in the future to hold a meeting in Springfield, and probably in other cities in the Commonwealth.

As you know, because of war conditions, it was found necessary to postpone the New England Postgraduate Assembly for the duration. This is very much regretted but, of course, cannot be helped. Another necessity was the transference of the annual meeting this year from Springfield, where it was scheduled to be held, back to Boston. However, your officers and the Committee on Arrangements felt that under the circumstances, because of gasoline restrictions and the probability that we would be able to entertain a larger number of doctors in Boston than in Springfield, this was desirable.

Another new committee that has performed an outstanding service is the Military Postgraduate Committee, organized the latter part of last year. This committee developed as the result of the simultaneous appreciation on the part of three of our members of the need for the Society of developing some plan of service for the various Army camps and hospitals, as well as Navy hospitals. There seemed to be a definite necessity for assisting the personnel of the camps, hospitals and stations to obtain speakers and to develop worth-while programs. The committee was organized in November, and since that time twenty-seven instructors have given thirty-two lectures at various Army installations and one at a Navy station. At a meeting of this committee the other evening, a gentleman representing the Navy voiced their appreciation of the advantages of this plan, and stated that they are going to adopt it and extend it throughout the Navy organization in the coming years.

This work has, of course, been carried on only by the Massachusetts Medical Society this year, but we believe that all the New England states should join in the program, since in practically all of them there are Army and Navy hospitals and stations that need assistance of this kind. Then too, the University of Vermont College of Medicine is situated in Burlington, the Yale University School of Medicine in New Haven, and I believe a two-year course is given by Dartmouth at Hanover. It should be a rather simple procedure to unite the New England states in this work. At the annual meeting of the Council last night, a motion was passed authorizing the Military Postgraduate Committee to continue its work and to get in touch with the other state societies through their secretaries, and our Society for the coming year was authorized to subsidize the expense of the program up to $500.

Another new and important committee is the War Participation Committee, this committee having been organized within the last two weeks. Its personnel consists of a progressive group of our members, and the committee has an excellent chairman. I am sure it will do some grand work during the war and for some years after the war is over.

Among other new activities of the Society that have developed during the year I now mention the action of the Council at its meeting last evening in adopting a resolution authorizing the officers of the Society to take over more space in the Boston Medical Library. I for one firmly believe that our home is and should be in the Boston Medical Library, since in my opinion these two organizations should work shoulder to shoulder. Situated as it is in the Fenway and so centrally located, it is easy of access from any part of Boston. As I have stated, the Society will very shortly take over more space. In view of several progressive movements now on foot, there is no question that the Society will use the present space and added space much more in the future than it ever has in the past. Plans are in progress for developing a medical service bureau, similar to the bureau maintained at the New York Academy of Medicine. This bureau will and should be the center of medical information in the City of Boston, having on record every day what is going on in the various hospitals and what medical meetings are taking place — in other words, making available up-to-the-minute information to any member of the Society or to any doctor concerning what is going on in and about Boston that will interest him. You will have more information on this project from time to time.

Another important resolution that was offered, voted on and accepted was the plan to establish a Postwar Loan

Fund. This subject was submitted to the Council by your president as follows:

The subject I am going to discuss with you is something that has been in my mind all year long, but it seemed to me that I should wait until the annual meeting of the Council before bringing it out. I shall present the plan I have in mind and shall ask Dr. Roger I. Lee, the incoming president, to exercise his prerogatives in appointing a committee.

The last war, as you know, was a comparatively short war. Many of us had been in practice five, ten or fifteen years when we went away. I happened to be in service for twenty-two months; some of us were in for ten, twelve or fourteen months—I believe the average was fourteen months. We got back into practice quickly. Some of us were lucky and had little trouble, but a lot of the men had difficulty getting started. I know because I talked with a great many of them. They needed a car, or they needed an office.

I can think of the story of one man, let's call him Bill Smith, who came back and was quite a hero. He had been wounded severely and was brought home, and when he came back he met John Jones, a banker, who thought Bill Smith was a wonderful hero and told him what a fine fellow he was. And then he said to Bill Smith, "What can I do for you?" Bill replied, "I should like to see if I can borrow a little money." The banker said, "Of course you can. I'll be glad to give it to you. How much do you want?" "Twelve hundred dollars." "By the way, what about collateral?" John Jones asked. Bill Smith said, "This will be only for a year." However, the banker looked at him with a cold eye and said, "If you haven't any collateral I can't let you borrow the money." That was poor Bill Smith's position. He was a great hero, he had fought for his country in the war and he had been wounded, but he never got the $1200—at least not from the banker.

A lot of men came back wounded and could not return to the type of practice they had engaged in before. They would have liked very much to have gone into something like refraction, but they did not have the money and they could not do it. As the result of the war many men lost out, not because of their service, but because of the fact they could not afford to rehabilitate themselves.

I think we have a great opportunity in the Massachusetts Medical Society today. This war is different. It is a long war. It has already lasted, so far as we are concerned, nearly a year and a half. It is going to last another two or three anyway. Our friends have gone, men who have been in practice five, ten or fifteen years. Many of those men are coming back wounded or in ill health. A surgeon may never be able to operate again. An internist may never be able to do that type of work. If he could do refraction or skin work, if he could get started with six or ten months' headway, it would be a great thing for him. A great many coming back are going to need special treatment until their wounds and their nerves are healed, and a majority of them are coming back without collateral.

My plan—and I have talked it over with a few men in the Society—is one to develop a fund in the Massachusetts Medical Society. Call it the Postwar Loan Fund. Let it run something on the order of the Morris Plan, any member of the Society being able, without embarrassment and without collateral, to make application for a loan. That application is endorsed by one or two members of the Society. The amount would be limited, and each application would receive careful consideration. Such a scheme could be easily put into effect. To come to us rather than to an otherwise affable banker, or to a loan shark, would save our friends and our colleagues a tremendous amount of embarrassment.

I can visualize a man coming home and wanting to go to Harvard or Boston University or Tufts for postgraduate study. Incidentally, possibly the medical schools might be willing to cut the fee in half or cut it out entirely for the ex-service man. The amount to be borrowed might be $1000 or $1500. That can be decided. The time in which the loan has to be repaid might run one or two years. That, too, would be decided by a committee.

I can visualize further. I can see men who are not coming back, who are leaving wives, and children who need to be educated. There are quite a number of paths this plan might follow.

You say, "Well, where are you going to get the money?" I think the preliminary fund could be obtained by assessing every member of the Massachusetts Medical Society who had not had the privilege of serving with the armed forces, because of age, disability or another reason, $10, with the opportunity of contributing more if he so desired. I think we could easily get $50,000 to start with, and if we needed more we have a backlog of over $200,000. Why does the Massachusetts Medical Society need $200,000 in bonds? That is something which has been a source of worry to me all year long. We have done nothing with it. It has just been invested. There must be something constructive we can do with the money instead of having it lie in our treasury.

A resolution was made, seconded, and accepted that the incoming President appoint a committee known as the Postwar Loan Fund Committee. This committee's duties will be to study every phase of this problem, to submit a report of progress at the meetings of the Council in October and in February, and to submit a final report at the annual meeting of the Council in May, 1944. You will, of course, be kept informed through your councilors with reference to this subject, and at the annual meeting next year have the final report of the committee.

As previously stated, the work of many other committees, standing and special, has been excellent and deserves the deepest appreciation of every member of the Massachusetts Medical Society. The reports of these committees will be incorporated in the report of the annual meeting of the Council, held on Monday evening, May 24, so that I feel it is unnecessary to mention them at this time, with the exception that I wish again to express to the chairmen and to the members of every committee in the Society my profound feeling of obligation and deepest appreciation for what they have done for me and for the Society during this rather trying year.

I have purposely held to the end of this report certain remarks with reference to the Medical Service Corporation, better known to you as the Blue Shield. You are all familiar, I am sure, with the fact that the Blue Shield

was inaugurated by the Massachusetts Medical Society, and was developed through the very excellent work of Dr. Thomas H. Lanman and Dr. James C. McCann. Dr. Lanman gave unselfishly of his time in obtaining from the Legislature the enabling act permitting us to organize this corporation. On his induction into the Army, it was necessary for him to relinquish the work, which was taken up by Dr. McCann and carried on so efficiently. May I say at this point that it is impossible for me to adequately express my appreciation of the work of Dr. Lanman, and my admiration for the work of Dr. McCann, in developing the Blue Shield and getting it where it is today. Dr. McCann has given tirelessly of his time and his talents, and the Massachusetts Medical Society will ever be indebted to him. It was my pleasure and privilege to make many trips with Dr. McCann to various parts of the country to study the different plans, and to determine the plan that would, in the opinion of Dr. McCann and his committee, work most efficiently in the Commonwealth of Massachusetts.

The Blue Shield began to function just about a year ago. The Blue Shield, gentlemen, in my opinion is a success, and as time goes on will be a greater success with each coming year. As a matter of fact, I am convinced that the Blue Shield will be one of the most successful projects the Massachusetts Medical Society has ever initiated and carried through. It is quite true that it has been a bit slow in getting under way, but this has been due to many factors beyond the control of the Blue Shield. One of the important reasons was the entrance of this country into the war; another, the development of multiple taxes, including the Victory tax. In my opinion by far the most important reason — and I say this with considerable diffidence and understanding — is that up to the present time the members of the Massachusetts Medical Society have on the whole not given the Blue Shield the united and loyal support that it should and must have. Unfortunately, many members have apparently made no effort to understand what the Blue Shield stands for, and many of these gentlemen have criticized the Blue Shield and made statements they otherwise would not have made, had they heard the clear exposition presented in practically every district medical society by Dr. McCann, and had they taken the time to read the several pamphlets sent to them and the articles appearing in the *New England Journal of Medicine*. It is my hope that in the ensuing year the members who have criticized and have not given their support to the Blue Shield will learn to understand it and will get back of it wholeheartedly. Only in this way will it be possible to make it the success that it should be.

At the present time there are forty groups enrolled in the Blue Shield; the total number of persons enrolled is 2329, with a total of 1170 contracts. Thirty-three hundred doctors have signed up as participating physicians, but again may I say that unfortunately not all this number have taken the time or given the effort to understand what the Blue Shield is, and what it will do for the individual and for them if given a fair trial. Frankly, it was our hope at this time last year that we would have approximately 3000 persons enrolled in the Blue Shield. As stated, we have 2329. With your support and cooperation we can, I believe, next year have 10,000 or more persons enrolled in the Blue Shield. May I earnestly beseech your understanding support of the Blue Shield. I am sure that, if there is any question that is not clear to any of you, merely writing to Dr. McCann, or if you wish writing to me, will bring you a prompt answer and clarification of the problem.

In concluding this report on the state of the Society, may I express to the members of the Massachusetts Medical Society the realization on my part of the great honor it has done me in permitting me to serve as president of the Society for the last year. I can assure you that I have kept the interest of the Massachusetts Medical Society paramount at all times and that I have done my best to see that the Society was conducted in a dignified manner during the year.

And, finally, I should like to leave with you a message that I have left with all the district societies that it has been my privilege to visit. It seems to me that a short quotation from a poem by Allan McIntosh, a young Scot who died in the last war, is most appropriate at this time — when all the world is in turmoil, when we are all concerned about our loved ones, and doing everything possible to bring about a quick victory and a lasting peace. Of the former, there is no question, for in my opinion victory is only a matter of time, and lasting peace a problem that we must and can solve. Allan McIntosh's lines appear on a very beautiful monument in Edinburgh — a memorial to the Scots who died in World War I. In bas-relief below the lines are shown Scottish pipers marching to war, their pipes playing, their scarfs blowing in the wind, and back of them the soldiers and back of them the artisans followed by the wives, the sweethearts and the children. Above this bas-relief appear these inspiring lines — lines, gentlemen, that I beg you to make your own as they are and have been mine for many years:

> If it be life that waits, I shall live forever unconquered;
> If death, I shall die at last strong in my pride — and free.

Dr. Schadt was greeted with loud applause at the close of his masterly address, which showed a wide knowledge of the problems facing the Society.

Dr. Schadt assumed the chair and introduced the annual orator, Dr. Edward P. Bagg, of Holyoke, chief medical evacuation officer of Region II and president of the staff of the Holyoke Hospital. Dr. Schadt called attention to the title of the oration — "Small Puddles." He wanted the members to understand that, even though Dr. Bagg was a pediatrician and therefore might be expected to know a good deal about small puddles, the reference in the title had nothing to do with Dr. Bagg's chosen field of endeavor.

The oration, which was published in the May 27 issue of the *New England Journal of Medicine* was beautifully and scholarly done, and Dr. Bagg was loudly applauded at its conclusion.

Dr. Schadt introduced Dr. Roger I. Lee, president for the ensuing year, Dr. Elmer S. Bagnall, president-elect, Dr. Eliot Hubbard, Jr., treasurer, and Dr. Michael A. Tighe, secretary. They all re-

sponded fittingly and expressed their pleasure at their enlarged opportunities to serve.

Dr. Frank R. Ober moved that the meeting adjourn. This motion was seconded by a member, and it was so voted.

Dr. Schadt declared the one hundred and sixty-second annual meeting of the Massachusetts Medical Society adjourned at 12:30 p.m.

MICHAEL A. TIGHE, *Secretary*

APPENDIX

ADMISSIONS RECORDED FROM MAY 26, 1942, TO MAY 25, 1943

YEAR OF ADMISSION	NAME AND RESIDENCE	MEDICAL SCHOOL
1942	*Abkowitz, Herbert, Everett	Kansas City University of Physicians and Surgeons
1942	*Adelman, Ernest Bernard, Bridgewater	Middlesex
1942	Albertowicz, Theodore John, Lowell	Tufts
1943	Allers, Olga Eleanore, Wellesley	Woman's Medical College of Pennsylvania
1942	Andreson, Lawrence Helmar, West Somerville	Tufts
1943	Angelo, Peter John, Quincy	Tufts
1942	Aranson, Albert, Newton Centre	Boston University
1942	*Argoff, Joseph, Fitchburg	Missouri College of Medicine and Science
1943	Avery, Bennett Franklin, Brookline	University of Michigan
1942	Baptista, Jesse, Fall River	Tufts
1943	*Berger, John Jacob, West Concord	Middlesex
1942	Berry, Franklyn Doane, New Bedford	Boston University
1942	*Bluestein, Jacob Kenneth, Bardsville	Middlesex
1942	*Boch, Louis S., Conway	Middlesex
1943	Boyd, James Gelston, Westborough	Boston University
1942	Brady, Frank William, Lowell	Tufts
1942	*Brahm, Leo, Worcester	Friedrich Wilhelm University of Berlin
1942	*Buoniconto, Pasquale, Hathorne	Royal University of Naples
1942	Burger, Franklyn Donaldson, Wellesley Hills	University of Michigan
1943	*Burns, Francis Albert, Foxboro	Middlesex
1942	Chace, John Frederick, Hudson	Tufts
1942	Clauser, William Joseph, Boston	Indiana University
1942	Cole, William Gregory, Pittsfield	Columbia University
1942	†Cook, William Wilder, Quincy	Tufts
1942	*Cooperstein, Harry Joshua, Fall River	Middlesex
1942	*Corrado, John Charles, East Boston	Middlesex
1942	Cosgrove, James Francis, Jr., Worcester	Tufts
1942	*Cottone, Peter Paul, Marlboro	Middlesex
1942	Cox, Robert Downing, Worcester	Tufts
1942	Crowell, Lois Blanche, Tewksbury	Boston University
1943	Currier, Wilber Dale, Brookline	University of Nebraska
1942	Curry, John James, Boston	Tufts
1942	Desmet, George William, North Andover	Tufts
1942	*Devine, Joseph William, Boston	College of Physicians and Surgeons, Boston
1942	*Donovan, William Francis, Watertown	Middlesex
1942	*Eames, Thomas Harrison, Arlington	College of Physicians and Surgeons, Boston
1943	Elliot, Howard Lailey, Brookline	McGill
1943	Epstein, Ida Florence, Roxbury	New York Medical College and Flower Hospital
1942	*Erbe, Henry Herman, Otis	Middlesex
1942	Fadgen, John Francis, Clinton	Loyola University
1942	Fanger, Herbert, Salem	New York Medical College and Flower Hospital
1942	*Ferrante, Anthony, Allston	College of Physicians and Surgeons, Boston
1942	Forsley, Richard, Lowell	Tufts
1942	Frank, Howard Alvin, Boston	New York University
1943	Gettings, Thomas Lawrence, Fall River	Tufts
1942	*Giallombardo, Nicholas Perrone, Somerville	Middlesex
1942	Gidman, Francis Thomas, Boston	Tufts
1942	Glass, Sarah Elizabeth, Worcester	Rush Medical College
1942	*Goglia, Alfred Anthony, Taunton	College of Physicians and Surgeons, Boston

1943	*Goldman, Harold Sumner, Medford	Middlesex
1942	*Goldstein, Morris, Cambridge	Middlesex
1942	Goodman, Doris Ryan, Natick	Tufts
1942	*Gordon, George Samuel, Lynn	Middlesex
1943	*Gordon, Robert Knott, Winthrop	Middlesex
1942	Grandfield, Robert Edward, West Roxbury	Harvard
1942	Grandmont, Irene Olivine, Foxboro	Boston University
1942	*Grzyb, Henry Andrew, Southbridge	College of Physicians and Surgeons, Boston
1942	Hardy, Erving Douglas, Worcester	Tufts
1942	Harken, Dwight Emary, Cambridge	Harvard
1942	Hayes, John Joseph, Jr., Brighton	Georgetown
1943	Hertig, Arthur Tremain, Newton Highlands	Harvard
1943	Hindle, William Vincent, Boston	Harvard
1943	*Hobica, Norman, Allston	Middlesex
1943	Howley, Edward Joseph, East Weymouth	Tufts
1943	Ingersoll, Francis McCall, Boston	Harvard
1942	Johnson, Carl Cordes, Brighton	Harvard
1942	*Johnson, Harry Taylor, Waltham	Middlesex
1943	Johnson, Walfred, Worcester	George Washington University
1942	*Kenney, John Aloysius, Mansfield	Midwest Medical School
1942	*Kenny, Donald Ellsworth, Medford	Middlesex
1942	Klam, Najeeb, Boston	Boston University
1943	Klemperer, Friedrich Wilhelm, Lexington	Harvard
1943	Liebman, Sumner David, Boston	Harvard
1942	*Limauro, Ulysses David, Lynn	Middlesex
1942	Loofbourow, Dorothea Gano, Cambridge	University of Cincinnati
1942	*Lowenberg, Benjamin, Dorchester	University of Lausanne
1942	MacMahon, Harold Edward, Cambridge	University of Western Ontario
1943	Mankowich, Ralph, Waltham	Boston University
1943	*Manning, Ralph A., Winchester	Middlesex
1943	McKell, David McCandless, Jr., Brookline	Harvard
1942	Miller, Edward Alexander, Sagamore	Boston University
1942	*Morrin, Harry Joseph, Roxbury	Middlesex
1942	*Nadelman, Benjamin Isidore, Roxbury	Middlesex
1942	Ober, Robert Emil, Cambridge	Tufts
1942	Olken, Harry George, Cambridge	Tufts
1942	Olshausen, Kenneth Wolfgang, Boston	University of California
1942	Porter, Reno Russell, Pepperell	Medical College of Virginia
1942	Potter, Theodore Alexander, West Newton	Boston University
1942	Quigley, George Edward, Newton	Boston University
1942	Rea, Stanley Leroy, Westfield	Stanford University
1943	Rice, Theodore Adams, Worcester	Harvard
1943	Richards, Anthony, Tewksbury	Tufts
1942	Rubin, Abraham Louis, Fort Devens	Tufts
1942	Rutledge, David Ivan, Wellesley Hills	University of Nebraska
1942	Ryan, William Francis, Palmer	Tufts
1942	*Sandler, Samuel, Cambridge	Middlesex
1942	Saward, Ernest Welton, Waltham	University of Rochester
1943	*Schiller, Louis, Pittsfield	Kansas City University of Physicians and Surgeons
1942	†*Schott, Norbert Abram, Wollaston	College of Physicians and Surgeons, Boston
1942	*Schwartz, Frank Harold, Fall River	College of Physicians and Surgeons, Boston
1943	*Schwartz, Leo, Springfield	Middlesex
1943	Seligman, Arnold Max, Brookline	Harvard
1943	*Shalett, Irving Joseph, Brookline	Middlesex
1943	*Sherman, David Edward, Springfield	Middlesex
1942	Shipp, Luther Murry, Medford	Tufts
1943	Silveus, Esther, Boston	University of Pittsburgh
1942	Sise, Herbert Stanwood, Brookline	Harvard
1942	Skinner, David, Newton Centre	Harvard
1942	*Smith, Leonard, Westfield	Middlesex
1943	*Solomon, Alex, Adams	Middlesex
1942	Sostek, Samuel Bertram, Medford	Boston University
1943	*Souza, Charles Marshall, Dighton	Middlesex
1942	*Sprinz, Helmuth, Boston	University of Berlin
1943	Stowell, Joseph May, Chestnut Hill	Tufts
1943	Suitor, Douglas Gilman, Springfield	Tufts
1942	Sullivan, Arthur James, Fall River	Jefferson Medical College
1942	*Sullivan, Francis Robert, Jamaica Plain	Middlesex

1943	Sullivan, Robert Edward, Dorchester	Tufts
1942	Taddeo, Arthur E., Natick	Tufts
1943	Tansey, Joseph Logan, Jamaica Plain	Harvard
1943	*Thannhauser, Siegfried Josef, Brookline	University of Munich
1942	Thorn, George Widmer, Cambridge	University of Buffalo
1942	*Tucci, John Hugh, Dorchester	Middlesex
1942	Tucker, Charles Albert, Boston	Tufts
1942	*Venti, Matthew, Methuen	Middlesex
1943	*Wallace, Thomas Edmund, Revere	Middlesex
1942	Waugh, Richey Laughlin, Brighton	University of Minnesota
1942	*Weinert, Thaddeus Conrad, Cambridge	Middlesex
1942	Williams, Conger, Milton	McGill
1942	Williams, John Webster, Newton	George Washington University
1942	*Wiseblatt, Aaron Carl, Dorchester	Middlesex
1942	*Wojcicki, Henry Telesfor, Fall River	Middlesex
1942	*Wyant, John Anderson, Fitchburg	College of Physicians and Surgeons, Boston
1942	Yasuna, Elton Ralph, Worcester	New York University
1943	Young, Edward Lorraine, III, Cambridge	Harvard
1943	*Zarella, Anthony George, Medford	Middlesex

Total number of new fellows admitted 136

*The candidate, after a personal interview, was approved by the Committee on Membership and permitted to take an examination before a board of censors.

†Deceased.

Deaths Reported from May 26, 1942, to May 25, 1943

ADMITTED	NAME	PLACE OF DEATH	DATE OF DEATH	AGE
1906	Adams, Charles Waldron	Cambridge	October 20, 1942	63
1895	†Ash, John Henry	Quincy	February 15, 1943	73
1899 } 1921 }	Bailey, William Thomas	Boston	January 16, 1943	74
1898	Blakely, David Newton	Brookline	October 15, 1942	75
1913	Bowen, Enos Emanuel	East Boston	July 15, 1942	60
1886	Brackett, Elliott Gray	Boston	December 28, 1942	82
1893	Briggs, Charles Albert	Assonet	December 14, 1942	79
1893	Bryant, Alice Gertrude	Boston	July 25, 1942	80
1899	Butler, Charles Shorey	Boston	February 23, 1943	72
1916	Butler, Francis Joseph	Worcester	June 7, 1942	59
1895	†Byrne, Charles Armstrong	Hatfield	April 3, 1943	80
1913	Carvell, Hanford	Lanesville	February 15, 1943	67
1891	†Chapin, Delia Lucretia	Springfield	October 7, 1942	88
1898	†Cochran, William James	Somerville	August 29, 1942	80
1942	Cook, William Wilder	Quincy	January 23, 1943	56
1913	Cox, Stanley Cullen	Holyoke	June 7, 1942	58
1906	Dana, Harold Ward	Brookline	May 8, 1943	65
1906 } 1923 }	Deems, Oren Manfred	Springfield	April 17, 1943	63
1896	Dennison, Archibald Sayre	Lynn	January 22, 1943	73
1881	Dixon, Robert Brewer	Boston	December 16, 1942	86
1899	Dow, David Crooker	Cambridge	May 27, 1942	67
1926	Dressler, Morris Lawrence	Fort Dix, New Jersey	May 6, 1943	54
1922	Dwyer, Philip Roche	Salem	February 28, 1943	49
1900	Easton, Elwood Tracy	Boston	January 31, 1943	67
1899	†Ellsworth, Samuel Walker	Quincy	November 27, 1942	72
1907	Faxon, William Otis	Stoughton	November 12, 1942	89
1911	French, Ralph Winward	Fall River	December 7, 1942	59
1895	†Gates, Ernest A.	Springfield	January 4, 1943	73
1926	Goldman, Harry	Roxbury	April 22, 1943	47
1888	Greenwood, Allen	Boston	October 23, 1942	76
1899	Handy, Harry Tucker	Scituate Center	October 8, 1942	68
1875 } 1921 }	†Harding, Edward Mitchell	Newton	December 19, 1942	90
1895	†Harris, Arthur Eugene	Lynn	July 11, 1942	72
1907	Henderson, Charles Russell	Reading	March 31, 1943	75

1903	Hickey, John Joseph	Peabody	January 17, 1943	75
1920	Hogan, Daniel John	Roslindale	August 15, 1942	55
1895	†Holmes, May Salona	Worcester	April 4, 1943	72
1887	†Jack, Edwin Everett	Brookline	November 16, 1942	79
1897	Jackson, Oliver Howard	Fall River	June 1, 1942	70
1915	Jacoby, Rudolph	Boston	August 20, 1942	52
1917	Jewett, Howard Wakefield	Lowell	March 18, 1943	63
1891	†Judkins, Frank Louville	Lynn	April 21, 1943	90
1890	†Kaan, George Warton	Sharon	April 14, 1943	88
1907 } 1927 }	†Kazanjian, Hampar Paul	Los Angeles	September 20, 1942	70
1932	Keeley, Thomas Henry	Monson	August 28, 1942	48
1892	†La Marche, Walter Joseph	Cambridge	May 30, 1942	80
1939	Laurin, Theophile	Lowell	April 10, 1943	72
1927	Leonard, Edward Joseph	Dorchester	February 15, 1943	41
1905	Lowell, William Holbrook	Winchester	March 31, 1943	67
1920	Lucy, John Joseph	Boston	December 28, 1942	47
1927	Luther, Eliot Horton	Westfield	May 2, 1943	47
1935	Maguire, James Alfred	Atlantic	May 31, 1942	36
1902 } 1939 }	Mahony, Francis Ronan	Lowell	May 17, 1943	64
1903	Malone, Charles	Jamaica Plain	October 10, 1942	67
1902	McBain, William Hearst	Malden	May 9, 1942	68
1933	McGuinness, John Francis	Woburn	June 17, 1942	49
1929	Metivier, Armand Oliva	Chicopee	July 1, 1942	52
1910	Miller, George Fremont	Belfast, Maine	March 22, 1943	67
1898	†Morse, Almon Gardner	Hingham	July 27, 1942	74
1928	Olef, Isadore	Dorchester	December 16, 1942	45
1889 } 1941 }	Peterson, Reuben	Duxbury	November 25, 1942	80
1910	Philbrick, Roscoe Hunter	New Orleans, Louisiana	Unknown	62
1911	Popoff, Constantine	Hampton Beach, New Hampshire	May 30, 1942	58
1901	Priest, Herbert Bancroft	Ayer	March 12, 1943	68
1926	†Rice, George Brackett	Brookline	March 28, 1943	83
1907	Robbins, Eugene Stanley	West Palm Beach, Florida	January 18, 1943	70
1942	Schott, Norbert A.	Boston	March 25, 1943	40
1897	Shultis, Frederick Charles	Leominster	June 23, 1942	69
1902	Stack, Charles Francis	Hyde Park	April 3, 1942	71
1892	Stickney, Edwin Pangman	Arlington	January 8, 1943	80
1909	Stone, Thomas Newcomb	Haverhill	April 21, 1943	63
1913	Temple, William Franklin	Boston	December 25, 1942	55
1937	Thau, William	Indiantown Gap Military Res., Pennsylvania	February 14, 1943	46
1908	Walker, Harry Abram	Somerville	October 30, 1942	61
1899	†Walsh, Charles Joseph	Gilbertville	September 6, 1942	83
1914	Willoughby, Earle Carlisle	North Reading	June 23, 1942	60

†Retired fellow.

Total number of deaths of active fellows	59
Total number of deaths of retired fellows	17
Grand total	76

Officers for 1943–1944

President: Roger I. Lee, Boston, 264 Beacon Street.

President-Elect: Elmer S. Bagnall, Groveland, 281 Main Street.

Vice-President: Daniel B. Reardon, Quincy, 1186 Hancock Street.

Secretary: Michael A. Tighe, Lowell. Office, Boston, 8 Fenway.

Treasurer: Eliot Hubbard, Jr., Cambridge, 29 Highland Street.

Assistant Treasurer: Norman A. Welch, West Roxbury. Office, Boston, 520 Commonwealth Avenue.

Orator: Joseph C. Aub, Boston, Massachusetts General Hospital.

Committees Elected by the Districts

Executive Committee of the Council — Established 1941 (Members *ex-officiis* and one councilor and alternate elected by the councilors of each district medical society).

President: Roger I. Lee, Boston, 264 Beacon Street.

President-Elect: Elmer S. Bagnall, Groveland, 281 Main Street.

Vice-President: Daniel B. Reardon, Quincy, 1186 Hancock Street.

SECRETARY: Michael A. Tighe, Lowell. Office, Boston, 8 Fenway.

TREASURER: Eliot Hubbard, Jr., Cambridge, 29 Highland Street.

Term Expires 1944

BERKSHIRE: Not yet elected.

FRANKLIN: Frederick J. Barnard, Greenfield, 479 Main Street. (Alternate: William J. Pelletier, Turners Falls, 113 Avenue A.)

HAMPDEN: George L. Steele, Springfield, 20 Maple Street. (Alternate: Edward A. Knowlton, Holyoke, 207 Elm Street.)

MIDDLESEX NORTH: William M. Collins, Lowell, 174 Central Street.

NORFOLK: Carl Bearse, Boston, 483 Beacon Street.

WORCESTER NORTH: C. Bertram Gay, Fitchburg, 62 Day Street. (Alternate: Francis A. Reynolds, Athol, 43 Cottage Street.)

Term Expires 1945

ESSEX SOUTH: Loring Grimes, Swampscott, 84 Humphrey Street. (Alternate: Charles L. Curtis, Salem, 10 Federal Street.)

HAMPSHIRE: L. Beverly Pond, Easthampton, 115 Main Street. (Alternate: Joseph D. Collins, Northampton, 187 Main Street.)

MIDDLESEX SOUTH: Dwight O'Hara, Waltham. Office, Boston, 416 Huntington Avenue. (Alternate: Sumner H. Remick, Waltham, 735 Trapelo Road.)

NORFOLK SOUTH: Daniel B. Reardon, Quincy, 1186 Hancock Street. (Alternate: Henry A. Robinson, Hingham, 205 North Street.)

SUFFOLK: Donald Munro, Boston, 818 Harrison Avenue. (Alternate: Charles C. Lund, Boston, 319 Longwood Avenue.)

WORCESTER: Ralph S. Perkins, Worcester, 10 Hackfeld Road. (Alternate: Gordon Berry, Worcester, 36 Pleasant Street.)

Term Expires 1946

BARNSTABLE: Not yet elected.

BRISTOL NORTH: William H. Allen, Mansfield, 70 North Main Street. (Alternate Ralph M. Chambers, Taunton, Taunton State Hospital.)

BRISTOL SOUTH: Not yet elected.

ESSEX NORTH: Frank W. Snow, Newburyport, 24 Essex Street. (Alternate: Rolf C. Norris, Methuen, 247 Broadway.)

MIDDLESEX EAST: Edward M. Halligan, Reading, 37 Salem Street. (Alternate: Richard Dutton, Wakefield, 33 Avon Street.)

PLYMOUTH: Peirce H. Leavitt, Brockton, 129 West Elm Street. (Alternate: Charles D. McCann, Brockton, 12 Cottage Street.)

Committee on Public Relations — Established 1931 (One councilor elected yearly by each district medical society; the president and president-elect of the Society are chairman and vice-chairman, respectively, and the vice-president and secretary of the Society are members *ex-officiis*).

BARNSTABLE: W. D. Kinney, Osterville.

BERKSHIRE: P. J. Sullivan, Dalton, 471 Main Street.

BRISTOL NORTH: J. H. Brewster, Attleboro, 178 South Main Street.

BRISTOL SOUTH: H. E. Perry, New Bedford, 159 Cottage Street.

ESSEX NORTH: E. S. Bagnall, Groveland, 281 Main Street (secretary of committee).

ESSEX SOUTH: Loring Grimes, Swampscott, 84 Humphrey Street.

FRANKLIN: W. J. Pelletier, Turners Falls, 113 Avenue A.

HAMPDEN: P. E. Gear, Holyoke, 188 Chestnut Street.

HAMPSHIRE: A. J. Bonneville, Hatfield, 60 Main Street.

MIDDLESEX EAST: J. H. Blaisdell, Winchester. Office, Boston, 45 Bay State Road.

MIDDLESEX NORTH: D. J. Ellison, Lowell, 8 Merrimack Street.

MIDDLESEX SOUTH: J. P. Nelligan, Cambridge, 2336 Massachusetts Avenue.

NORFOLK: N. A. Welch, West Roxbury. Office, Boston, 520 Commonwealth Avenue.

NORFOLK SOUTH: F. A. Bartlett, Wollaston, 308 Beale Street.

PLYMOUTH: C. D. McCann, Brockton, 12 Cottage Street.

SUFFOLK: A. A. Hornor, Boston, 319 Longwood Avenue.

WORCESTER: J. M. Fallon, Worcester, 390 Main Street.

WORCESTER NORTH: J. G. Simmons, Fitchburg, 30 Myrtle Avenue.

Committee on Legislation — Established 1942 (One councilor elected yearly by each district medical society).

BARNSTABLE: Not yet elected.

BERKSHIRE: P. J. Sullivan, Dalton, 471 Main Street.

BRISTOL NORTH: J. L. Murphy, Taunton, 23 Cedar Street.

BRISTOL SOUTH: Not yet elected.

ESSEX NORTH: E. H. Ganley, Methuen, 251 Broadway.

ESSEX SOUTH: C. A. Worthen, Lynn, 19 Park Street.

FRANKLIN: H. M. Kemp, Greenfield, 42 Franklin Street.

HAMPDEN: W. A. R. Chapin, Springfield, 121 Chestnut Street.

HAMPSHIRE: A. N. Ball, Northampton, Northampton State Hospital.

MIDDLESEX EAST: J. M. Wilcox, Woburn, 6 Bennett Street.

MIDDLESEX NORTH: W. H. Sherman, Lowell, 9 Central Street.

MIDDLESEX SOUTH: B. F. Conley, Malden, 51 Main Street (chairman of committee).

NORFOLK: J. C. V. Fisher, West Roxbury. Office, Boston, 510 Commonwealth Avenue.

NORFOLK SOUTH: D. L. Belding, Hingham. Office, Boston, 80 East Concord Street.

PLYMOUTH: J. J. McNamara, Brockton, 231 Main Street (interim appointment).

SUFFOLK: W. B. Breed, Boston, 264 Beacon Street.

WORCESTER: L. M. Felton, Worcester, 36 Pleasant Street (secretary of committee).

WORCESTER NORTH: F. A. Reynolds, Athol, 43 Cottage Street.

Committee on Nominations — Established 1874 (One councilor and alternate elected yearly by each district medical society).

BARNSTABLE: W. D. Kinney, Osterville. (Alternate: P. M. Butterfield, Harwich.)

BERKSHIRE: P. J. Sullivan, Dalton, 471 Main Street. (Alternate, C. F. Fasce, Pittsfield, 311 North Street.)

BRISTOL NORTH: W. H. Allen, Mansfield, 70 North Main Street. (Alternate: J. L. Murphy, Taunton, 23 Cedar Street.)

BRISTOL SOUTH: E. F. Cody, New Bedford, 105 South 6th Street. (Alternate: R. B. Butler, Fall River, 278 North Main Street.)

ESSEX NORTH: G. L. Richardson, Haverhill, 94 Emerson Street. (Alternate: R. C. Norris, Methuen, 247 Broadway.)

ESSEX SOUTH: D. S. Clark, Salem, 2 Oliver Street. (Alternate: P. P. Johnson, Beverly, 1 Monument Square.)

FRANKLIN: F. J. Barnard, Greenfield, 479 Main Street. (Alternate: W. J. Pelletier, Turners Falls, 113 Avenue A.)

HAMPDEN: A. G. Rice, Springfield, 146 Chestnut Street. (Alternate: E. P. Bagg, Holyoke, 207 Elm Street.)

HAMPSHIRE: L. B. Pond, Easthampton, 115 Main Street. (Alternate: J. D. Collins, Northampton, 187 Main Street.)

MIDDLESEX EAST: R. R. Stratton, Melrose, 538 Lynn Fells Parkway. (Alternate: E. M. Halligan, Reading, 37 Salem Street.)

MIDDLESEX NORTH: M. A. Tighe, Lowell, 9 Central Street. (Alternate: W. H. Sherman, Lowell, 9 Central Street.)

MIDDLESEX SOUTH: Dwight O'Hara, Waltham. Office, Boston, 416 Huntington Avenue. (Alternate: J. C. Merriam, Framingham, 198 Union Avenue.)

NORFOLK: D. D. Scannell, Jamaica Plain. Office, Boston, 475 Commonwealth Avenue. (Alternate: H. J. Inglis, Chestnut Hill. Office, Boston, 43 Bay State Road.)

NORFOLK SOUTH: D. B. Reardon, Quincy, 1186 Hancock Street. (Alternate: H. A. Robinson, Hingham, 205 North Street.)

PLYMOUTH: W. H. Pulsifer, Whitman, 26 Park Avenue. (Alternate: P. H. Leavitt, Brockton, 129 West Elm Street.)

SUFFOLK: W. B. Breed, Boston, 264 Beacon Street. (Alternate: A. A. Hornor, Boston, 319 Longwood Avenue.)

WORCESTER: R. P. Watkins, Worcester, 332 Main Street. (Alternate: W. F. Lynch, Worcester, 390 Main Street.)

WORCESTER NORTH: B. P. Sweeney, Leominster, 5 Gardner Place. (Alternate: G. P. Keaveny, Fitchburg, 62 Fox Street.)

STANDING COMMITTEES FOR 1943-1944

ELECTED BY THE COUNCIL, MAY 24, 1943

Date of Appointment

PUBLICATIONS — Established 1825.

R. M. Smith	June 6, 1933 (appointed chairman May 21, 1941)
J. P. O'Hare	June 9, 1936
Conrad Wesselhoeft	June 2, 1937
W. B. Breed	February 7, 1940
Oliver Cope	May 21, 1941

ARRANGEMENTS — Established 1849.

G. M. Morrison	May 21, 1941 (appointed chairman June 24, 1942)
R. J. Heffernan	May 25, 1942
S. C. Wiggin	June 24, 1942
R. I. Smith	June 24, 1942
G. G. Bailey	November 13, 1942

ETHICS AND DISCIPLINE — Established 1871.

R. R. Stratton	June 9, 1936 (appointed chairman May 21, 1941)
W. J. Brickley	February 3, 1937
A. G. Rice	June 1, 1938
F. R. Jouett	May 21, 1940
A. R. Gardner	May 21, 1941

MEDICAL EDUCATION — Established 1881.

R. T. Monroe	May 21, 1941 (appointed chairman February 4, 1942)
G. D. Henderson	June 1, 1938
C. S. Keefer	February 4, 1942
I. R. Jankelson	May 25, 1942
A. W. Allen	May 24, 1943

MEMBERSHIP — Established 1897.

H. F. Newton	June 9, 1931 (appointed chairman May 25, 1942)
John E. Fish	June 17, 1930
P. H. Leavitt	June 1, 1938
A. W. Reggio	May 21, 1940
S. H. Remick	May 24, 1943

W. H. Allen, H. Q. Gallupe, A. E. Parkhurst — representing the Supervising Censors.

PUBLIC HEALTH — Established 1912.

F. P. Denny	June 1, 1938 (appointed chairman June 7, 1939)
G. N. Hoeffel	June 17, 1930
H. L. Lombard	June 4, 1935
H. F. Day	June 7, 1939
F. W. Marlow, Jr.	May 25, 1942

MEDICAL DEFENSE — Established 1927.

A. W. Allen	June 7, 1927 (appointed chairman June 7, 1939)
E. D. Gardner	June 7, 1927
W. R. Morrison (sec. *pro tem.*)	June 9, 1936
Horatio Rogers	June 7, 1939
I. M. Dixson	August 17, 1942

SOCIETY HEADQUARTERS — Established 1932.

W. H. Robey	February 24, 1937 (chairman)
C. G. Mixter	June 8, 1932
E. C. Miller	June 4, 1935
G. L. Schadt	May 24, 1943
M. A. Tighe	May 24, 1943

FINANCE — Established 1938.

F. C. Hall (interim appointment)	July 8, 1943 (chairman)
E. L. Hunt	June 2, 1938
C. F. Wilinsky	June 2, 1938
E. J. O'Brien, Jr.	June 2, 1938
P. P. Johnson	October 4, 1939

INDUSTRIAL HEALTH — Established 1942.

Dwight O'Hara	May 25, 1942 (chairman)
J. C. Aub	May 25, 1942
D. L. Lynch	May 25, 1942
H. C. Marble	May 25, 1942
T. L. Shipman	May 25, 1942
J. N. Shirley	May 25, 1942
F. N. Manley	May 24, 1943

SPECIAL COMMITTEES

CANCER — Established 1917.

E. L. Hunt, *chairman*; F. G. Balch, E. M. Daland, P. E. Truesdale, C. C. Simmons.

REPRESENTATIVES TO THE MASSACHUSETTS CENTRAL HEALTH COUNCIL

BARNSTABLE: W. D. Kinney
BERKSHIRE: R. J. Carpenter
HAMPDEN: G. D. Henderson
NORFOLK: F. P. Denny
SUFFOLK: R. B. Osgood
WORCESTER: E. C. Miller

PUBLIC EDUCATION (a subcommittee of the Committee on Public Health) — Established 1930.

F. P. Denny, *Chairman*; G. N. Hoeffel, *secretary*; G. R. Minot, W. H. Robey, E. H. Place, C. C. Simmons, J. H. Pratt, H. W. Stevens, J. B. Ayer, H. P. Mosher (interim appointment), F. R. Ober, E. P. Joslin, J. D. Barney, H. L. Lombard, Joseph Garland.

POSTGRADUATE INSTRUCTION — Established 1932.

Reginald Fitz, *chairman*; L. E. Parkins, *secretary*; J. W. O'Connor, R. N. Nye, C. J. Kickham.

PHYSICAL THERAPY — Established 1935.

A. L. Watkins, *chairman*; F. P. Lowry, R. B. Osgood.

COMMITTEE TO CONSIDER EXPERT TESTIMONY — Established 1936.

F. R. Ober, *chairman*; David Cheever, F. P. McCarthy, Carl Bearse, W. J. Brickley.

COMMITTEE ON AUTOMOBILE INSURANCE CLAIMS — Established 1937.

H. C. Marble, *chairman*; H. M. Landesman, *secretary*; P. P. Henson.

COMMITTEE ON CONVALESCENT CARE — Established 1938.

T. D. Jones, *chairman*; H. E. Gallup.

COMMITTEE TO STUDY THE PRACTICE OF MEDICINE BY UNREGISTERED PERSONS — Established 1939.

Richard Dutton, *chairman*; B. F. Conley, E. F. Timmins.

TWENTY-FIVE VOTING MEMBERS IN MASSACHUSETTS HOSPITAL SERVICE, INC. — Established 1939.

B. H. Alton, E. S. Bagnall, G. M. Balboni, W. B. Breed, L. D. Chapin, H. F. Day, J. F. Donaldson, A. W. Dudley, John Fallon, J. E. Flynn, A. R. Gardner, H. W. Godfrey, J. H. Lambert, A. A. Levi, J. C. Merriam (interim appointment), Donald Munro, A. E. Parkhurst, Helen S. Pittman, A. G. Rice, A. T. Ronan, F. W. Snow, G. L. Steele, R. R. Stratton, J. E. Talbot, E. L. Young.

COMMITTEE CONCERNED WITH PREPAYMENT MEDICAL-CARE COSTS INSURANCE — Established 1940.

J. C. McCann, *chairman*; E. S. Bagnall, P. H. Leavitt, G. L. Schadt, J. C. Merriam (interim appointment).

COMMITTEE ON TAX-SUPPORTED MEDICAL CARE — Established 1940.

E. S. Bagnall, *chairman*; A. L. Duncombe, A. A. Hornor, E. L. Hunt, W. J. Pelletier.

COMMITTEE TO MEET WITH THE MASSACHUSETTS HOSPITAL ASSOCIATION — Established 1940.

W. G. Phippen, *chairman*; Frederic Hagler, E. D. Gardner, A. E. Parkhurst, F. W. Snow, E. A. Adams.

COMMITTEE ON MATERNAL WELFARE — Established 1941.

J. A. Smith, *chairman*; Thomas Almy, R. L. DeNormandie, Eoline C. Dubois, C. J. Duncan, M. F. Eades, A. F. G. Edgelow, Florence L. McKay, J. W. O'Connor, L. E. Phaneuf, G. M. Shipton, W. R. Sisson, R. M. Smith, R. S. Titus, R. J. Williams.

COMMITTEE ON REHABILITATION — Established 1941.

W. E. Browne, *chairman*; W. M. Collins, J. J. Regan, B. F. Andrews, R. M. Chambers, A. L. Watkins, John Fallon.

COMMITTEE CONCERNED WITH POSTPAYMENT MEDICAL-CARE COSTS THROUGH BANKS — Established 1942.

E. S. Bagnall, *chairman*; H. G. Stetson, D. J. Ellison.

COMMITTEE TO AID THE BOSTON MEDICAL LIBRARY — Established 1942.

W. H. Robey, *chairman*; David Cheever, M. A. Tighe, Eliot Hubbard, Jr., C. E. Mongan.

COMMITTEE TO MEET WITH THE MEDICAL ADVISORY COMMITTEE OF THE INDUSTRIAL ACCIDENT BOARD — Established 1942.

D. J. Ellison, *chairman*; G. M. Morrison, D. D. Scannell.

MILITARY POSTGRADUATE COMMITTEE — Established 1942.

W. R. Ohler, *chairman*; L. E. Parkins, *secretary*; C. S. Keefer, S. H. Proger, F. R. Ober, G. M. Morrison (interim appointment).

COMMITTEE ON WAYS AND MEANS TO CONSERVE PHYSICIANS' ENERGIES — Established 1942.

E. S. Bagnall, *chairman*; C. F. Wilinsky, J. J. Dumphy.

WAR PARTICIPATION COMMITTEE — Established 1943.

W. B. Breed, *chairman*; R. R. Stratton, *vice-chairman*; Reginald Fitz, Dwight O'Hara, M. A. Tighe, Carl Bearse.

COMMITTEE TO AID THE DISTRICT RATIONING ADMINISTRATOR — Established 1943.

Joseph Garland, *chairman*; F. G. Brigham, F. W. White (all interim appointments).

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REPRESENTATIVE TO MENTAL HEALTH FOR DEFENSE ORGANIZATION.

Abraham Myerson.

REPRESENTATIVE TO THE HOSPITAL COUNCIL OF BOSTON FOR THE YEAR 1943.

F. H. Colby.

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June 1, 1942, to June 1, 1944	
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Dwight O'Hara, Waltham	E. L. Hunt, Worcester
C. E. Mongan, Somerville	C. J. Kickham, Brookline
W. G. Phippen, Salem	J. I. B. Vail, Hyannis
June 1, 1943, to June 1, 1945	
A. G. Rice, Springfield	P. J. Sullivan, Dalton
R. H. Miller, Boston	John Fallon, Worcester

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(This is not a committee of the Massachusetts Medical Society. It is here listed for purposes of information only.)

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J. I. B. Vail, Hyannis.
D. H. Hiebert, Provincetown.

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C. C. Persons, New Bedford.
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CASE RECORDS OF THE MASSACHUSETTS GENERAL HOSPITAL

Weekly Clinicopathological Exercises

FOUNDED BY RICHARD C. CABOT

TRACY B. MALLORY, M.D., *Editor**

BENJAMIN CASTLEMAN, M.D., *Acting Editor*

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CASE 29291

PRESENTATION OF CASE

A fifty-three-year-old housewife entered the hospital because of fluid in the right chest.

Approximately three months prior to admission, following a cat scratch on the right thigh, she developed a tender red swelling of the thigh that spread in the next three days to the knee and upper calf. Simultaneously an "erysipeloid" reaction developed on the medial aspect of the left upper thigh. Her physician treated her with sulfadiazine by mouth for a week and the inflammation subsided. However, several days later the entire right leg became warm, swollen and mottled, and two or three days later the left leg assumed a similar appearance. After five weeks of bed rest most of the infection subsided. Three and a half weeks prior to admission, the patient became progressively short of breath, and ten days later at a community hospital 900 cc. of bloody fluid were withdrawn from the right pleural cavity. She felt better after the tap and returned home. Three days prior to admission marked breathlessness returned, and since the pleural fluid had reaccumulated, she was referred to this hospital for further care. During the illness her temperature usually ranged from 99.4 to 100°F., but on two occasions at night it rose to 103. There was no hemoptysis, chills, weight loss or night sweats.

The family and past histories were noncontributory.

Physical examination revealed a somewhat obese, slightly dyspneic woman who coughed occasionally but did not raise any sputum. The chest was large, and the anteroposterior diameter increased. There was limitation of expansion of the chest, particularly on the right. Flatness was percussed over the right chest posteriorly as high as the scapula. In this area the breath and voice sounds were inaudible. Anteriorly over the right apex and posteriorly over the left lower chest and just above the area of flatness on the right there were showers of fine crackling rales, principally on expiration, that did not clear after coughing. A blowing systolic murmur was heard over the entire precordium but best in the tricuspid area. There was moderate tenderness to deep palpation in both lower quadrants of the abdomen, and there was a sense of resistance in the lower midabdomen; no definite masses were felt. The right liver edge was percussed four fingerbreadths below the costal margin. Over both knees, the lateral aspect of the right ankle and the left calf there was a macular, bright-red, smooth rash. There was no edema. Homans's sign for thrombophlebitis was negative.

The blood pressure was 175 systolic, 95 diastolic. The temperature was 101.6°F., the pulse 112, and the respirations 31.

The red-cell count was 4,950,000, and a stained smear showed 64 per cent neutrophils. The urine was normal. A blood Hinton test was negative. The protein was 4.2 gm., the sugar 108 mg., and the nonprotein nitrogen 33 mg. per 100 cc.

An electrocardiogram showed normal sinus rhythm, with a rate of 115. The PR interval was 0.16 second. The T wave was low in Leads 1, 2 and 3, and inverted in Lead 4; there was slight right-axis deviation.

An x-ray film of the chest showed a moderate amount of fluid and air in the right pleural cavity that collapsed the right lung to about half its usual size. The fluid rose to the level of the third rib anteriorly. The right half of the diaphragm was elevated and showed considerable limitation in motion. There was some increase in the markings of the left lung field; otherwise this lung was not remarkable. The heart and mediastinum were slightly displaced to the left. The carina was not widened. The right main bronchus was not sufficiently well visualized to determine intrinsic disease. X-ray films and laminograms after removal of 1200 cc. of bloody xanthochromic fluid from the right chest revealed an apparent mass at the right hilus that extended into the right upper lobe.

During hospitalization the patient seemed quite short of breath but had few complaints. The temperature was usually 100°F.; the pulse ranged from 80 to 125, and the respirations from 20 to 45.

On the twelfth hospital day the patient suddenly began to sweat profusely and the pulse became rapid and thready. She denied pain. The cardiac apical rate was 120, and there was a prominent gallop rhythm. Respirations were 35, and the systolic blood pressure was 80. The extremities were cold and clammy. She was treated supportively and seemed somewhat better. Half an hour later the left arm became blue, painful and cold and no brachial or radial pulse could be felt. She failed rapidly, became cyanotic and dyspneic, and died five minutes later.

*On leave of absence.

Differential Diagnosis

Dr. Chester M. Jones: To me this history outlines a very curious sequence of events. It is obvious that the patient had some sort of infection in the lower extremities. She subsequently developed a bloody pleural effusion. At no time did she have pain in the chest, which is one of the striking things in the story. Three months after the onset of her first symptoms, she was admitted to the hospital, still with fluid in the chest, but at that time there was no swelling of the legs, although it had been present about two months previously. There still was, however, evidence of an inflammatory process described as an erythematous rash. The chest fluid was first bloody and later bloody and xanthochromic, indicating that blood had been there for a long time and that some of the hemoglobin had changed to bile pigment. I assume that the pneumothorax developed when air was introduced during the chest tap. Such a finding complicates the picture somewhat. Then with the terminal event, which came on suddenly, again there was no pain. If one states these facts in sequence — infection of the right thigh, something that was called erysipelas, subsequently warmth, swelling and mottling of the lower legs, first on the right and then on the left, and finally something in the chest — it is reasonable to say that this woman had infection, then thrombophlebitis in the deep veins of the legs, probably the superficial femoral vein, and finally pulmonary embolism.

There are several queer things, however, about this diagnosis. She never had any pain, either in the legs or in the chest. Furthermore, two months after the first swelling and redness appeared in the legs, we are told that she had no further swelling and the Homans's sign for phlebitis was negative. That does not prove there was not a deep phlebitis. Certainly we have seen numerous cases in which there was thrombophlebitis productive of a pulmonary embolus and in which we were unable to recognize the thrombophlebitis on the basis of physical signs in the leg. It is still more curious that when examined here there was no demonstrable edema, although more than once I have seen pulmonary embolism due to phlebitis of the deep veins of the leg that was not accompanied by demonstrable edema. It is not the thing to expect, however.

I should like to see the x-ray films of the chest at this time and raise the question whether the apparent mass described is suggestive of a primary bronchiogenic carcinoma. If so, we may be dealing with several diagnoses here instead of one. The collapse of the lung, apparently due to air and fluid in the pleural cavity, explains why the diaphragm was high. After removal of a lot of fluid the lung was still collapsed, apparently because the diaphragm was very high.

Dr. Laurence L. Robbins: The diaphragm is high, but I doubt that it has anything to do with the air that is in the pleural cavity. Usually with collapse of the lung due to fluid and air in the pleural cavity the lung is compressed mechanically, and under these circumstances one would expect the right half of the diaphragm to be low.

Dr. Jones: Yes; but if there is compression against the collapse of the lung and compression of a bronchus that might cause a high diaphragm rather than a low one.

Dr. Robbins: Usually that happens when there is no fluid or air.

Dr. Jones: Do you think it is correct to say that the diaphragm is high?

Dr. Robbins: Yes.

Dr. Jones: The laminograms show something definite?

Dr. Robbins: Yes. Although they are not satisfactory in the usual sense, because we cannot see the outline of the right main bronchus or the trachea, they show a definite line in the area that should represent the base of the right upper lobe, with partial consolidation or something invading it. The shadow in the region of the hilus suggests enlarged lymph nodes. In this film it has a lobulated margin.

Dr. Jones: If there were a bronchiogenic carcinoma producing collapse of a portion of the lung, and a mass at the hilus, would you not expect a slightly different picture?

Dr. Robbins: Not necessarily. If this is a bronchiogenic carcinoma, I should say that it was located fairly close to the right upper lobe division, that is, in one of the secondary bronchi of the right upper lobe, and that the hilar mass was probably metastatic or direct extension of the tumor.

Dr. Jones: The thing that amazes me is the relatively painless and insidious occurrence of the bloody fluid in the pleural cavity. The absence of hemoptysis, pain and sudden onset in the early part of the story is I believe rather striking clinical evidence against pulmonary embolism. Furthermore, bloody fluid is quite unusual in the presence of pulmonary infarction unless the infarct is very large, or close to the pleural surface, in which case it is surprising there was no pain. I believe that a bloody pleural effusion can follow a peripheral infarction, but this sequence of events is rather unusual with the sort of story that this patient presented.

Dr. Benjamin Castleman: All bland infarcts extend to the pleural surface of the lung, and

only about 15 per cent of them are directly associated with pleural fluid, which may or may not be hemorrhagic.

DR. JONES: On the basis of the x-ray findings, it seems to me that this is not the picture one would expect with a pulmonary infarction or multiple pulmonary emboli. From the story I am more inclined to link the hilar lesion, which is definite in the x-ray film, with a malignant process, and I believe that bronchiogenic carcinoma is the most logical diagnosis. If that is so, it explains the bloody pleural fluid. I think it is fair to say that in addition to everything else, this patient had hypertensive heart disease and presumably was asthmatic. I think the cause of death may well have been an acute pulmonary embolus or coronary thrombosis. However, the rapidity with which the symptoms ensued makes me think that the terminal episode was associated with pulmonary embolism.

The electrocardiogram is going to be difficult to interpret. There was definite displacement of the heart when the electrocardiogram was taken It had moved appreciably to the left, but the striking thing is, even with displacement of the heart to the left, that the electrocardiogram is recorded as showing right-axis deviation, when one would expect left-axis deviation. But in spite of the right-axis deviation, there was a low T wave in the first three leads and inversion of the T wave in Lead 4. That I believe is entirely consistent with right-sided heart failure and not at all inconsistent with pulmonary embolism. There was heart failure, as evidenced by the large liver, and it was undoubtedly right sided — let us say a cor pulmonale as a result of something going on in the chest. One of the conditions in which right-sided heart failure develops rather acutely is in association with pulmonary emboli.

I cannot make a diagnosis with any degree of assurance, but I believe that the patient had a thrombophlebitis of the lower extremities and I should not be at all surprised if there were pulmonary emboli. I also believe there was a terminal pulmonary embolism, together with right-sided heart failure. The systolic murmur over the tricuspid area is interesting; I think it was due to an incompetent or insufficient tricuspid valve and not necessarily to endocarditis. The mass in the mediastinum is probably better explained on the basis of malignancy than of anything else.

DR. JACOB LERMAN: How do you explain the final episode of arterial embolus to the left arm unless you assume endocarditis?

DR. JONES: That was a curious thing. It was noted distinctly as a terminal event, and I do not know how to interpret it. Half an hour after the sudden collapse, the left arm was noted to be cold, blue and so forth. Of course one can say there was an embolic occlusion of the brachial artery associated with acute circulatory collapse. The systolic blood pressure dropped from 175 to 80.

A PHYSICIAN: How about something in the pulmonary veins? I know such lesions are rare.

DR. CASTLEMAN: Thrombi in the smaller pulmonary veins occur probably a lot more frequently than we realize.

A PHYSICIAN: I was thinking of embolus from the main pulmonary veins.

DR. CASTLEMAN: I have never seen one except as an extension from a mural thrombus in the left auricle or an extension from subacute bacterial endocarditis of the mitral valve.

DR. REED HARWOOD: I should like to mention the possibility of paradoxical embolism, part going through a septal defect to produce occlusion of the brachial artery.

CLINICAL DIAGNOSES

Carcinoma of lung.
Embolus of left brachial artery.

DR. JONES'S DIAGNOSES

Thrombophlebitis of legs.
Pulmonary embolism.
Acute cor pulmonale.
Bronchiogenic carcinoma?
Hypertensive heart disease.

ANATOMICAL DIAGNOSES

Adenocarcinoma of lung, right upper lobe, with metastases to bronchial, mesenteric and retroperitoneal lymph nodes, pulmonary lymphatics, liver, pleura and peritoneum.
Acute massive pulmonary embolism.
Phlebothrombosis of legs.
Subacute cor pulmonale.
Pulmonary infarct, left lower lobe.
Hemohydrothorax, right.
Hydrothorax, left.

PATHOLOGICAL DISCUSSION

DR. CASTLEMAN: On opening the right pleural cavity a 1500 cc. of hemorrhagic, slightly turbid fluid was found, and scattered along the parietal pleura were numerous flecks of obvious carcinoma. The primary cancer was in the main bronchus of the right upper lobe, about 3 cm. from the carina. There were large metastatic bronchial lymph nodes, which represented the shadow seen in the right hilus. There were metastases throughout the liver, and this adequately explains the size of the liver. There were also metastases to the peritoneum and to some of the mesenteric and retroperitoneal lymph nodes. Microscopically the

tumor was a poorly differentiated adenocarcinoma. The left pleural cavity contained about 50 cc. of straw-colored fluid, and in the left lower lobe was a small hemorrhagic infarct of several days' duration. The immediate cause of death was a massive embolism to the main pulmonary artery, which apparently arose from thrombi in the deep veins of the calf of one leg. The veins of both legs were thrombosed. Most of the lymphatics throughout both lungs were plugged with carcinoma, and I believe this was the cause of the right-axis deviation. This is the second case of this condition, called "subacute cor pulmonale" by Brill and Robertson,[1] that we have recently seen.[2] I am sure that the infarct in the left lower lobe was not enough to have produced right-sided heart failure.

We examined the right subclavian and axillary veins and arteries but were unable to find an embolus.

References

1. Brill, I. C., and Robertson, T. D. Subacute cor pulmonale. *Arch. Int. Med.* 60:1043-1057, 1937.
2. Case records of the Massachusetts General Hospital (Case 29281). *New Eng. J. Med.* 229:123-126, 1943.

CASE 29292

Presentation of Case

A thirty-two-year-old unmarried woman, an office worker, entered the hospital complaining of nausea and soreness in the right abdomen.

Five months prior to admission, after a brisk walk, the patient noted the onset of crampy pain in the right lower quadrant, which doubled her up and caused nausea without vomiting. For two days following the attack she had tenderness in the right lower quadrant. Her physician found a soft movable mass under the umbilicus, and marked tenderness in the right lower quadrant. The uterus was small and anteflexed. In the right vault was a tender, large, thick, cystic mass. Three days later the mass had disappeared, although there was still marked tenderness, as well as a hard tender mass, in the right lower quadrant. The patient was not seen for the next four months; during that period she had several similar attacks of crampy pain, which doubled her up and forced her to go to bed. During some attacks she felt nauseated and had either diarrhea or constipation. The last menstrual period began thirteen days prior to admission, and associated with this the patient had an attack of severe pain in the right lower quadrant and lower abdomen that lasted several hours. After a three-day menstrual flow the pain disappeared and she then resumed normal activity. During the past six months the patient had lost 10 pounds.

The family history was noncontributory. The menarche occurred at fourteen; periods were regular, with a twenty-eight-day interval, and lasted three days, four or five napkins being used a day. There was moderate crampy pain in the lower abdomen during the first day, but no clots were passed and there was no leukorrhea. Menstrual cramps had increased in severity during the two years prior to admission. Six years prior to admission, at a community hospital, a uterine suspension, appendectomy and resection of an ovarian cyst were performed.

Physical examination disclosed a well-developed and well-nourished woman. The heart and lungs were normal. There was a well-healed midline scar in the lower abdomen. Deep in the right lower quadrant there was a slightly tender, fairly indefinite sausage-shaped mass. Pelvic examination revealed a small uterus and cervix. The whole right vault was occupied by a large, tender, thick-walled mass.

The blood pressure was 110 systolic, 75 diastolic. The temperature, pulse and respirations were normal.

Examination of the blood revealed a hemoglobin of 11.6 gm. per 100 cc. The urine was normal. A blood Hinton test was negative.

An operation was performed on the second hospital day.

Differential Diagnosis

Dr. Francis Ingersoll: It is of interest to note that this patient's pain was always on the right side, was always described as being crampy and was always associated with nausea without vomiting and that she had had increasing dysmenorrhea over a period of two years. The record does not state that the ovary was removed, so we can assume that she still had a portion of the right ovary. On physical examination there was a mass in the right lower quadrant and one in the pelvis. One cannot tell whether the same mass was felt by pelvic and abdominal examination.

This was an acute abdominal case, and it seems to me that the story of having had crampy abdominal pain with nausea makes one consider in the differential diagnosis some pathologic process that involved the gastrointestinal tract. On the other hand, since the pain seemed to come on with her periods, one must also consider a disease involving the genital tract. One might naturally assume that something was wrong with the appendix. However, since the appendix had been removed, it need not be considered in the differential diagnosis. Although it is often true that a patient is told

that the appendix has been removed when it has not, I think that we can discuss this case with the assumption that it was.

If one considers a pathologic process involving both the genital tract and the gastrointestinal tract, the first one that comes to mind is endometriosis. Cases have been reported in which the patient had endometriosis in the pelvis and endometriosis involving the bowel, usually around the sigmoid on the left side. Endometriosis may even involve the small bowel to such an extent that the patient has some degree of intestinal obstruction. So, as the first and best possibility to explain this patient's symptoms I should say that she had endometriosis, that the thick-walled cyst that was felt in the pelvis was an endometrioma, and that she also had an endometriosis that involved the small bowel. Possibly the small bowel was adherent to the endometrioma in the pelvis, and when she had her last episode of pain, which was with a period, she had bled into the endometrioma.

There are many other possibilities that should be considered. The patient might very well have had chronic pelvic inflammation. Certainly in pelvic inflammatory disease a person can have many gastrointestinal symptoms and often pain associated with periods. That might explain the symptoms, but it seems to me that with pelvic inflammatory disease the patient would not have been so comfortable, and she should have had more fever at some time during her illness. Although the episode five months prior to admission might have been the time that she had her first attack of pelvic infection, why a cystic mass was felt and later disappeared is hard to explain by pelvic inflammatory disease. This can, however, be explained by endometriosis. On the other hand, this might have been some type of bilateral ovarian cyst. Possibly the mass was an ovarian cyst that had twisted at the time of the first attack and had caused the pain after she had taken a walk. At the time she had the pelvic mass there was a mass in the right lower quadrant, so one would have to postulate that she had two ovarian cysts to account for the physical findings. One might explain the syndrome on the basis of regional enteritis, although that is just a stab in the dark. Certainly nausea and crampy pain can be caused by this disease, but one would expect some fever with it. An inflamed Meckel's diverticulum could produce a mass in the pelvis and something that could be felt in the right lower quadrant. However, the best explanation that I can offer is endometriosis, an endometrioma in the right vault to account for the gastrointestinal symptoms and endometriosis involving the small bowel.

Dr. Benjamin Castleman: Have you anything to add, Dr. Meigs?

Dr. Joe V. Meigs: I think that Dr. Ingersoll has discussed the case exceedingly well, and I agree with him.

Dr. Castleman: Perhaps you can answer the question about the umbilical mass, Dr. Simmons?

Dr. Fred Simmons: The mass was described on the first office visit, and disappeared following a soapsuds enema three days later. I think that can rightly be termed a "red herring." I followed this woman for a month; she then disappeared and I had to write a letter to get her in again. The mass was still present, and since I could not rule out ovarian tumor, I believed that she needed exploration for that reason. Endometriosis was certainly a likely possibility.

On opening the abdomen we found an ovarian tumor on the right. The ovary was three times normal size, the upper third being replaced by a large cyst, which was believed to be endometriosis. The rest of the ovary was normal. Both tubes and ovaries were bound down by multiple, firm, dense adhesions, apparently the result of endometriosis. Adherent to the right tube and ovary was a greatly dilated ptotic type of cecum, which overlay the broad ligament and the back of the right cornu of the uterus. The rectum was adherent to the left tube and uterus. All the adhesions were broken up by sharp and blunt dissection, and the cecum was freed from the adnexa and uterus. I assumed that the first operation was for endometrioma in the right ovary and that many of the adhesions were due to the earlier operation. The patency of the right tube was demonstrated. Because the patient was thirty-two years old and because in this hospital the trend is toward conservative surgery in endometriosis for the sake of later childbearing, only the large cyst in the right ovary was resected, the uterus being suspended. She made an uneventful convalescence.

Clinical Diagnoses

Ovarian cyst.
Pelvic inflammatory disease.

Dr. Ingersoll's Diagnosis

Endometriosis of ovary, pelvis and small intestine.

Anatomical Diagnosis

Endometriosis of ovary and pelvis.

Pathological Discussion

Dr. Castleman: The ovarian cyst showed characteristic endometriosis without evidence of inflammation around it.

DR. MEIGS: We see a lot of endometriosis at this hospital. Do you see it at the Free Hospital for Women, Dr. Rock?

DR. JOHN ROCK: Yes; it is a common disease.

DR. MEIGS: It seems to me that endometriosis is a physiologic process and occurs in almost every woman who does not have children. Perhaps that is the answer; perhaps it is not. In a large percentage of the patients I operate on, if the pelvis is examined carefully an area of endometriosis will be found.[1] It is a disease, and it is pathologic when it is far enough advanced to interfere with normal function. When I find small areas of endometriosis I consider them to be of academic interest, and remove a piece for diagnosis. The percentage of endometriosis in my operative cases must be about 40 per cent. It is a condition that occurs in women who do not have children and is usually of little significance.

DR. CASTLEMAN: In what percentage would you say that it does not involve the ovary?

DR. MEIGS: A large percentage. It is frequently found in uterosacral ligaments and on the bladder flap near its junction with the uterus.

DR. CASTLEMAN: Do you believe that Sampson's[2] theory of the pathogenesis of endometriosis is ruled out by not finding it in the ovary?

DR. MEIGS: Sampson's work is so good that it is hard to deny it. I have a feeling that his explanation is correct for certain cases. We had a patient recently who had both tubes shut off by inflammatory disease for ten years, yet she had endometriosis in the uterosacral region.

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A HOLMES CENTENNIAL

No prophet lacks honor, save in his own country. The centennial of Oliver Wendell Holmes's justly celebrated paper, "The Contagiousness of Puerperal Fever," published in the *New England Quarterly Journal of Medicine and Surgery* in April, 1843, was scarcely noted in Boston, though it received distinguished attention elsewhere. It was a noteworthy article, for it first introduced into America the doctrine of the infective nature of puerperal septicemia. Doctor Irving's address, published elsewhere in this issue of the *Journal*, makes honorable amends for the apparent neglect, and pays deserved tribute to the man who dared raise his voice in the wilderness to point out the responsibility of physicians for the spread of childbed fever.

The fame of Doctor Holmes, however, remains securely enshrined. His memory is freshly treasured in the Department of Anatomy at the Harvard Medical School, where he taught for so many years, and where many of his felicitous phrases are still repeated in lectures. As "Autocrat of the Breakfast Table" he diffused his genial humor and philosophy through the columns of the *Atlantic* to a much wider circle. Some of his poems, like "The Chambered Nautilus," are immortal. His character and personality are typical of the best in Boston, where for years he dwelt as *genius loci* of the Back Bay. Even a few are still alive who remember seeing him, in frock coat and silk hat, walking up Beacon Hill, and stopping to contemplate

> The spendthrift crocus bursting through the mould
> Naked and shivering with his cup of gold.

Anatomist, autocrat, poet, Oliver Wendell Holmes has left his soul on earth, and, like the *biformis vates* Horatius Flaceus, whom he so much resembled, he is double lived in Heaven too.

THE PLUMMER—VINSON SYNDROME

In 1922, Vinson,[1] of the Mayo Clinic, reported on his observations of 69 patients having inability to swallow as a prominent and sometimes only symptom. The paper was entitled "Hysterical Dysphagia." Fifty-seven of his patients were females, twelve were males. Patients with so-called "globus hystericus" were excluded from the series.

Vinson noted that the dysphagia usually began suddenly, often when the patient choked on a piece of solid food, the point of obstruction being high in the throat. This was followed by inability to swallow solid food, particularly meat and green vegetables, as well as difficulty in swallowing pills or capsules. Liquids and soft solids were usually well taken, and the loss of body weight was not marked. There did develop, however, a moderate

or secondary anemia, which Vinson thought was due to the quality of the food taken. In 37 of the patients the hemoglobin was 60 per cent or under. Enlargement of the spleen was noted in 12 cases in which the history of dysphagia was of long standing and the anemia, for the most part, was marked. Roentgenographic examinations in all cases gave negative results; and esophagoscopic examinations failed to reveal any lesions.

Although a plain esophageal sound was passed into the stomach in all cases, this resulted in no real stretching of the esophagus, and the effective treatment was on the basis of suggestion. This was followed by encouragement in eating solid foods and by the administration of iron and arsenic. Vinson states that the blood picture improved rapidly, the spleen returned to normal size and usually nothing further was needed to bring about an immediate, temporary cure. He found that recurrences were likely unless the patients were constantly reassured in regard to their condition. Sometimes a second passage of the sound was needed.

Vinson added that Plummer first pointed out this complex of dysphagia without organic obstruction, anemia and enlargement of the spleen. No reference, however, was given to Plummer's account, and it presumably was not published.

In the discussion of Vinson's paper, which was presented before the Minnesota State Medical Association on August 26, 1921, Dr. R. Rizer, of Minneapolis, suggested that Vinson's name should be given to this disease. Another discusser did not agree with this point of view, but in spite of his opposition, the name "Plummer—Vinson Syndrome" came into the literature and has received wide recognition.

It is now known that this syndrome was described before 1922, when Vinson's paper was published. Hurst[2] has recently called attention to the fact that the syndrome was noted by Paterson[3] at a meeting of the Laryngological Section of the Royal Society of Medicine, held on May 2, 1919. Paterson gave an even more complete description of the condition than did Vinson, for he noted atrophy of the mucous membrane of the tongue, which is now known to accompany this condition. At the same meeting, this syndrome was also described by Kelly.[4] He, too, noticed that in a few cases the mucous membrane of the pharynx had a pale, waxy aspect and that the tongue was smooth and devoid of papillae. Kelly attributed this appearance to the anemia resulting from the restricted and often insufficient diet. Hurst does not refer to this paper by Kelly, which seems to have equal value with that by Paterson. It is Hurst's opinion that the condition should be known as "the upper-dysphagia-with-anemia syndrome of Paterson." To this should be added the name of Kelly. It would be even more advisable to use the term suggested by Suzman,[5] "the syndrome of anemia, glossitis and dysphagia."

Hurst states that the syndrome occurs in about 15 per cent of all cases of simple achlorhydric anemia, is usually noted between the ages of thirty and fifty and is rarely found in men. It appears to be the direct result of iron deficiency due to a diet containing too little meat and green vegetables. The anemia is rapidly overcome by the administration of 30 gr. of iron and ammonium citrate three times a day. In some long-standing cases, malignant degeneration has followed the atrophy of the mucous membrane.

A good many facts about this syndrome are not clear, and differentiation from allied conditions is not always possible. Strictly speaking, the dysphagia is due to a functional loss in the first stage of swallowing, in moving the food from the anterior part of the mouth back to the pharyngeal wall, where the reflex is initiated. The swallowing itself, when the bolus reaches this point, is normal, and thus the condition is not demonstrable by fluoroscopy with barium. The syndrome should therefore not be confused with many conditions that result in difficulty in swallowing in the second stage, such as constriction of the pharynx and esophagus due to scars or tumor and paralysis of the pharyngeal muscles occurring temporarily in myasthenia gravis and permanently in bulbar paralysis. There may, however, be anemic condi-

tions of the mucous membrane, pharynx or esophagus that lead to dysphagia of another type. These cannot be classed as examples of the Plummer–Vinson syndrome, at least if one adheres to the original descriptions. Such a confusing condition was described in the January 28 issue of the *Journal*.[6] In that case, in spite of the retention of a mouthful of barium in the hypopharynx and the pyriform sinuses owing to what appeared to be a paralysis of the pharynx, with narrowing of the cervical esophagus, all of which led to a diagnosis of carcinoma of the esophagus, at post-mortem examination no explanation for the pharyngeal paralysis or for the esophageal constriction was found.

In spite of uncertainty in regard to the exact nature of this syndrome, it seems clear, in view of the work of Paterson and Kelly, that the name "Plummer–Vinson syndrome" should not be retained and that, if any names are to be connected with it, Paterson and Kelly should be honored. By whatever name this syndrome is designated, however, the condition itself needs further study from the laryngologist, the internist and the pathologist.

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MEDICAL EPONYM

Foramen of Winslow

This entity was described by Jacques-Bénigne Winslow (1669–1760) in *Exposition anatomique de la structure du corps humain* [*An Anatomical Exposition of the Structure of the Human Body*] (Paris, 1732). The following quotation appears on pages 171 and 172 of the second volume of the English translation by G. Douglas (London, 1743):

> We see from this situation of the two omenta that, in the space left between the lower side of the stomach and upper side of the mesocolon, they have a very broad communication with each other; so that if either of them contained in its cavity, any fluid, that fluid might readily get between the stomach and mesocolon and so pass into the other bag; especially when the stomach is empty and consequently its situation easily changed.
>
> Therefore, by means of this interstice between the stomach and mesocolon, the two omenta form one cavity, which opens into the cavity of the abdomen by one common orifice situated near the commissure on the right side of the great omentum. This orifice is semilunar or semicircular, and formed by the union of two membranous ligaments, whereof one connects the beginning of the duodenum and neck of the vesicula fellis to the liver; the other connects the contiguous portion of the colon to the same viscus, and extends to the pancreas. From thence arises an incurvated border which surrounds the root of the lobulus, leaving an opening wide enough to admit the end of the finger.
>
> To discover this orifice of the omentum, we need only raise a little the great lobe of the liver and find out the root of the lobulus and apply to it a large pipe wrapt round with cotton, wool or tow, to hinder the regress of the air. Then if we blow gradually, the air will inflate the sides of the great omentum and give it the appearance of a large bladder irregularly divided into several lobes or tubercles by the bands of fat which appear in this state like so many fraena between the lobes.

R. W. B.

MASSACHUSETTS MEDICAL SOCIETY

DEATH

GERSTEIN — Maurice Gerstein, M.D., of Brookline, died July 13. He was in his seventy-fourth year.

Dr. Gerstein received his degree from New York University Medical College. He was chief surgeon at the Jewish Memorial Hospital, Roxbury, and was on the staff of the Beth Israel Hospital.

He was a member of the Massachusetts Medical Society and the American Medical Association.

His widow and three daughters survive.

WAR ACTIVITIES

CIVILIAN DEFENSE

New Recommendations for Treatment of Burns and Prevention of Wound Infections

The Medical Division of the Office of Civilian Defense, Washington, D. C., has revised its pamphlet *Treatment of Burns and Prevention of Wound Infections* to incorporate new technics that have been developed within the past year. The recommendations in this pamphlet are based on recent directions of the Committee on Chemotherapeutic and Other Agents and the Subcommittee on Burns of the Committee on Surgery, Division of Medical Sciences, National Research Council. Originally drawn up by these committees for the armed forces, the recommendations have been modified to adapt them to the problems involved in the treatment of civilian casualties.

Recommendations for the use of sulfonamides are accompanied by the observation that these drugs must be used more cautiously in the treatment of civilian wounds

than is necessary in the care of military casualties, for the following reasons:

> The injured may include individuals of all ages and with various types of pre-existing disease, instead of a selected group of healthy young males. The possibility of toxic effects is therefore greatly enhanced. Moreover, it is assumed that in civilian injuries, hospitalization will be possible in a relatively short time, whereas in military operations such is not always the case. This usually makes it possible to postpone all consideration of chemotherapy until the injured have been hospitalized. It is then possible to administer sulfonamides with better safeguards and to consider such contraindications as other pathological conditions or known sensitivity to individual drugs. The dangers of dehydration can also be better prevented or overcome under such circumstances.

In a discussion of intra-abdominal wounds leading to perforation of the hollow viscera, the revised pamphlet advises sodium sulfadiazine as the drug of choice for parenteral administration, which is considered preferable to oral therapy during the first forty-eight hours. Sulfanilamide was recommended in the previous edition. Concentrated solutions of sodium sulfadiazine are not recommended for subcutaneous or intramuscular routes, but it is pointed out that weak (0.5 per cent) solutions may be used with little danger of sloughing of the tissues.

Special emphasis is placed on the danger of giving sulfonamide drugs to a patient who is not voiding normally (over 1000 cc. per day). The pamphlet warns:

> Should circumstances require sulfonamide administration in the presence of inadequate urinary output, the urine should be watched for evidence of renal damage and the dosage of drug adjusted so that a blood concentration, as evidenced by daily determinations, not to exceed 10 mg. per 100 cc., is maintained. . . . If further diminution of the urinary output occurs, administration of the drug should be stopped immediately and fluids should be forced orally, if possible, and by means of glucose and water (5 per cent in sterile distilled water) intravenously if necessary. If anuria due to bilateral obstruction of the ureters develops, ureteral catheterization and lavage of the renal pelves may be required.

The emergency care of burns is outlined as follows:

> Whenever casualties with extensive burns can be admitted to hospitals without delay, and definitive treatment can be instituted promptly, morphine sulfate, ½ gr., should be administered at the scene of the incident and no local therapy applied to the burned area except sterile gauze to exposed surfaces to prevent infection.

The most notable change in the OCD pamphlet is the withdrawal of the recommendation of the use of ointments or jellies containing tannic acid in the first-aid treatment of burns. The new advice given is that when definitive care cannot be carried out within two hours, the patient should receive sufficient morphine to relieve pain (not less than ½ gr., except in patients with lung and bronchial [and cerebral] damage, the very old or the very young); and the burned surfaces should be covered with sterile boric acid ointment or petrolatum over which one or two layers of gauze of fine mesh (44) is smoothly applied. Over this dressing thick sterile gauze or sterile cotton waste is placed and the entire dressing is bandaged firmly but not tightly. Substitution of jelly containing 5 per cent sulfathiazole in water-soluble base, which is supplied by the OCD Carrying-Case A for mobile medical teams, is permissible.

The discussion of definitive treatment of burns has been expanded to stress the necessity for administration of large amounts of plasma:

> In patients with severe burns, quantities up to 12 units or more may be required in the first twenty-four hours. . . . To the patient in critical condition, plasma must be given rapidly (as much as 500 cc. in ten minutes may be necessary) and not allowed to flow drop by drop. It must never be administered by any other than the intravenous route. Syringe injection may be used. If facilities for hematocrit determinations are available, the following general rule can be used for guidance regarding the amount of plasma required. For each point that the hematocrit is above 50 per cent cells, at least 100 cc. of plasma should be administered. If clinically satisfactory results are not obtained with this dosage, larger quantities should be given.

A footnote points out that rapid administration of intravenous fluids may be dangerous to cardiac patients and that the physician's judgment will have to determine the amount as well as the rate of administration in such cases.

The pamphlet describes open and closed treatment for burns. The open treatment that is now considered the treatment of choice and is especially recommended for the treatment of burns of the hands, face, feet, perineum and genitalia, consists essentially of the application of boric acid ointment or petrolatum, with pressure dressings. Such dressings can often be left in place twelve or fourteen days. The closed treatment, which is the tanning or eschar method, is particularly indicated in extensive "flash" or second-degree burns of the trunk. This method is recommended only if the following conditions are present: if not more than twenty-four hours have elapsed; if the burned area has not been grossly contaminated; if strict surgical asepsis is employed in the preparation of the burned surface; and if coagulation is rapidly accomplished, that is, by the combined use of tannic acid and silver nitrate. The method of tanning described is the same as that given in the original edition of the pamphlet.

In the new directions, additional emphasis is placed on masking of both the patient and his attendants, to minimize the danger of secondary infection.

NEW HAMPSHIRE MEDICAL SOCIETY

DEATH

LAMPREY — ALICE CHESLEY LAMPREY, M.D., of Exeter, died June 30. She was in her eighty-second year.

Dr. Lamprey received her degree from Tufts College Medical School in 1900. She was a member of the New Hampshire Medical Society.

Her husband and a sister survive.

MISCELLANY

MASSACHUSETTS DEPARTMENT OF PUBLIC HEALTH

RÉSUMÉ OF COMMUNICABLE DISEASES IN MASSACHUSETTS FOR MAY, 1943

DISEASE	MAY 1943	MAY 1942	SEVEN-YEAR MEDIAN
Anterior poliomyelitis	1	2	1
Chicken pox	1124	1763	1207
Diphtheria	4	16	11
Dog bite	1156	1335	1259
Dysentery, bacillary	12	5	5
German measles	8080	2443	259
Gonorrhea	360	321	358
Measles	6990	5320	4118
Meningitis, meningococcal	94	17	8
Meningitis, other forms	18	11	–
Meningitis, undetermined	8	1	–
Mumps	645	2017	977
Pneumonia, lobar	232	200	386
Salmonella infections	6	3	20
Scarlet fever	1949	1120	923
Syphilis	507	523	507
Tuberculosis, pulmonary	303	321	303
Tuberculosis, other forms	19	23	30
Typhoid fever	1	2	4
Undulant fever	4	1	4
Whooping cough	537	970	757

Meningococcal meningitis, while still at a figure nearly twelve times the seven-year median and still surpassing all records antedating March of this year, is dropping decidedly, with only 94 cases reported, compared with last month's 140 and the previous month's 130 cases.

Thus far, 1943 has seen scarlet fever at a notably high level. Since March, 1941, the monthly figures for this disease have been consistently higher than those of the previous year. In 1942, the increase became marked. The highest point in thirty-seven years was reached in April of the current year. During May, however, a significant drop is apparent, the monthly figure being the lowest since January. As regards distribution of cases, it is about as one would expect, in that the bulk of the cases occurred east of Worcester in the area of greatest population. Springfield, however, has had an unusual number of cases of scarlet fever, which reached a peak in March and has been dropping sharply since then.

GEOGRAPHICAL DISTRIBUTION OF CERTAIN DISEASES

Anterior poliomyelitis was reported from: Boston, 1; total, 1.

Anthrax was reported from: Haverhill, 1; total, 1.

Diphtheria was reported from: Fall River, 1; Lowell, 1; Melrose, 1; Somerville, 1; total, 4.

Dysentery, bacillary, was reported from: Boston, 8; Medford, 4; total, 12.

Encephalitis, infectious, was reported from: Leicester, 1; Needham, 1; Waltham, 1; total, 3.

Malaria was reported from: Camp Edwards, 1; Fort Banks, 8; Fort Devens, 2; New Bedford, 1; total, 12.

Meningitis, meningococcal, was reported from: Arlington, 1; Attleboro, 1; Auburn, 1; Bellingham, 1; Belmont, 1; Billerica, 2; Boston, 30; Bridgewater, 1; Brookline, 2; Cambridge, 2; Camp Edwards, 7; Chelsea, 2; Charlton, 1; Easthampton, 1; Fairhaven, 1; Fall River, 1; Falmouth, 1; Fort Banks, 1; Fort Devens, 2; Framingham, 1; Hanover, 1; Haverhill, 1; Holyoke, 1; Hudson, 1; Lawrence, 1; Leominster, 1; Lowell, 2; Malden, 3; Melrose, 1; Methuen, 1; Milford, 1; Milton, 1; New Bedford, 1; Newburyport, 1; Quincy, 3; Revere, 1; Rockport, 1; Somerville, 1; Springfield, 3; Webster, 1; Williamstown, 2; Woburn, 1; Worcester, 4; total, 94.

Meningitis, other forms, was reported from: Belmont, 1; Bourne, 1; Boston, 3; Cambridge, 1; East Bridgewater, 1; Easthampton, 1; Haverhill, 1; New Bedford, 1; Newton, 1; Norwood, 1; Quincy, 1; Walpole, 1; Westfield, 1; Worcester, 3; total, 18.

Meningitis, undetermined, was reported from: Arlington, 1; Newton, 1; Spencer, 1; Springfield, 2; Wilmington, 1; Worcester, 1; Wrentham, 1; total, 8.

Salmonella infections were reported from: Everett, 1; Fall River, 1; Lawrence, 2; Natick, 1; North Andover, 1; total, 6.

Septic sore throat was reported from: Boston, 7; Brookfield, 1; Haverhill, 1; Merrimac, 1; total, 10.

Tetanus was reported from: Lynn, 1; total, 1.

Trachoma was reported from: Fall River, 1; Weymouth, 1; total, 2.

Typhoid fever was reported from: Dalton, 1; total, 1.

Undulant fever was reported from: Boston, 1; Gloucester, 1; Uxbridge, 1; Williamstown, 1; total, 4.

BOOK REVIEWS

The Medals of the United States Army Medical Department and Medals Honoring Army Medical Officers. By Edgar E. Hume, M.D., D.P.H., D.T.M., LL.D. 16°, paper, 169 pp., with 23 plates. New York: The American Numismatic Society, 1942. $3.00.

Colonel Hume, a well-known historian of the Medical Department of the United States Army, has listed all the medals that have been issued honoring army medical officers. With each description of the medal itself is contained a brief note about the individual who established the gift and the man honored by the medal. There is also considerable historical information in regard to the founding of the Medical Department of the United States Army and the establishment of the Army Medical School, the Army Veterinarian School, the Medical Field Service School and other departments, including the Army School of Nursing and the School of Aviation Medicine. Each medal is illustrated.

This record is invaluable as a historical document and contains considerable material not easily available in other form. The book is a complete, carefully documented account, which should be of interest to a wide audience and should serve as a reference book in every medical library. Medical historians and librarians are greatly indebted to the American Numismatic Society for making this monograph possible.

The Answer Is . . . Your Nerves. By Arnold S. Jackson, M.D. With a chapter by The Rev. Edwin O. Kennedy. 12°, cloth, 197 pp., with 10 illustrations. Madison, Wisconsin: Kilgore Printing Company, 1942. $2.00.

This is a popular account of nervous diseases as seen in a large clinic and also in private practice. The book is well written and adequately covers the field. Of particular value are the striking and often amusing illustrations that accompany the text. It is a useful, small publication, which should be widely read, both by physicians and their patients.